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OPHTHALMOLOGY

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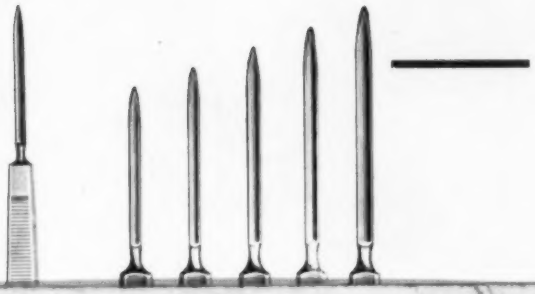

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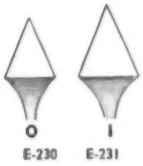

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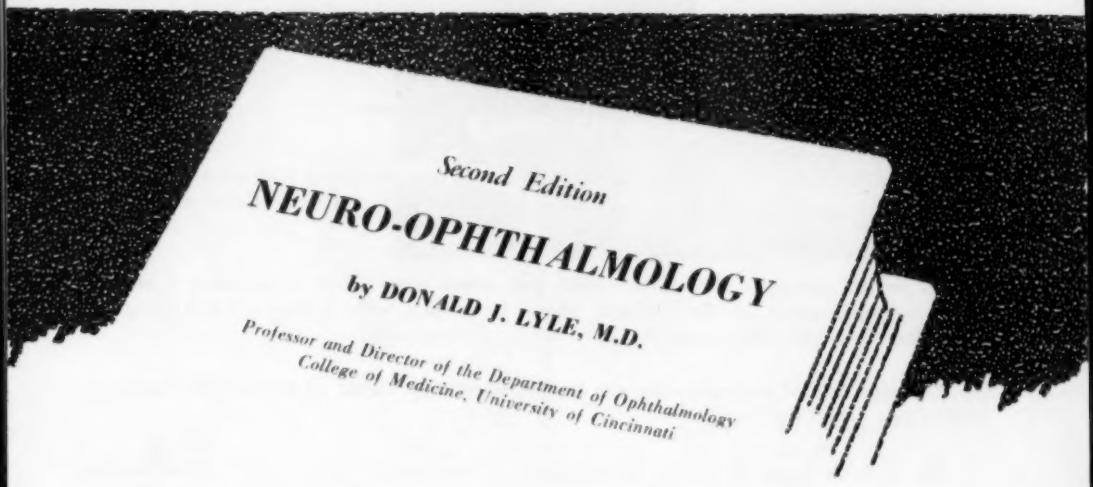
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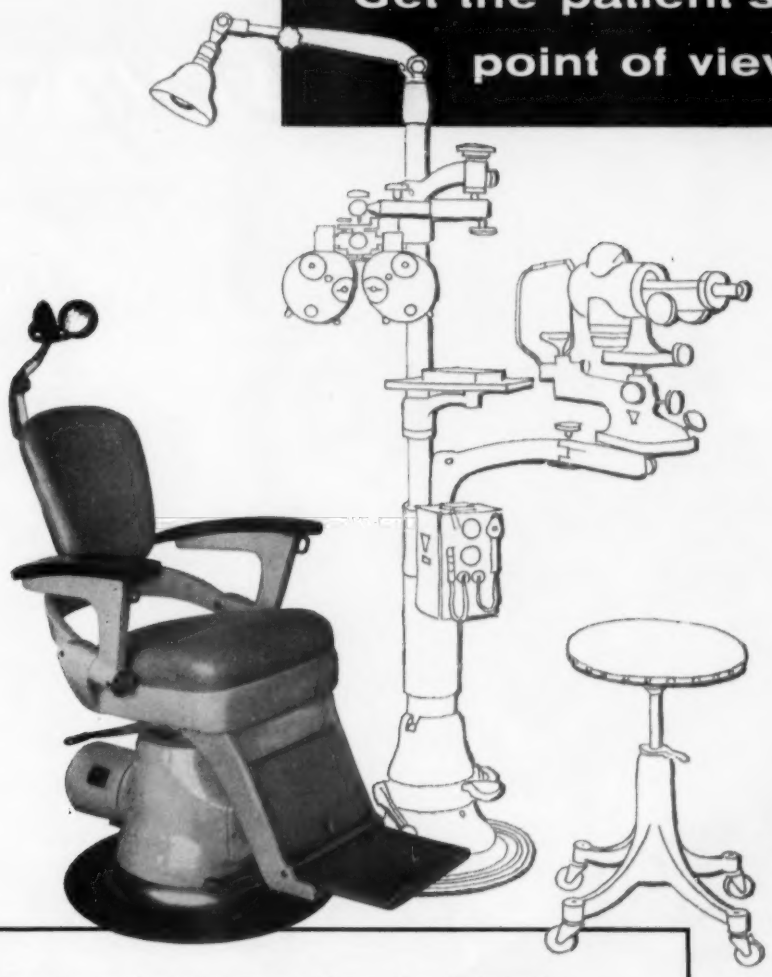

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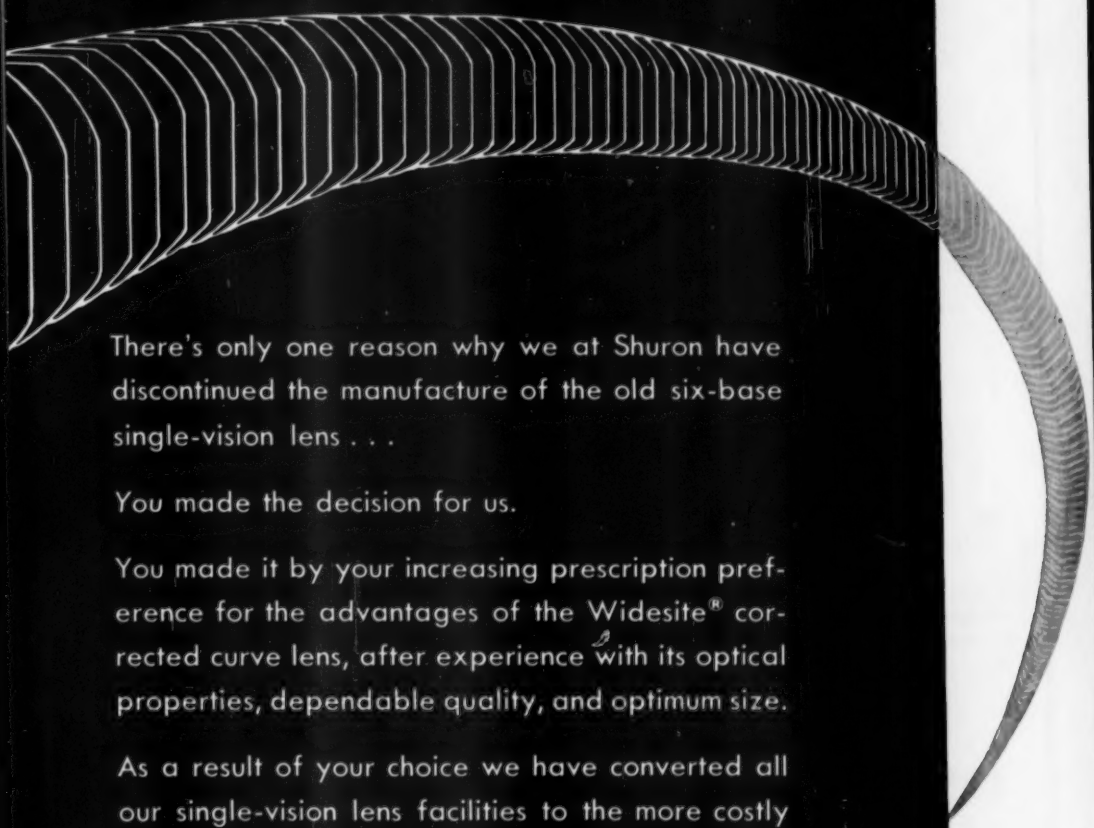

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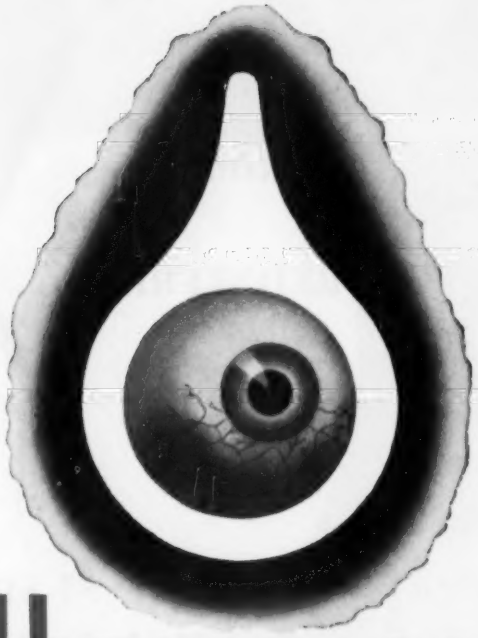

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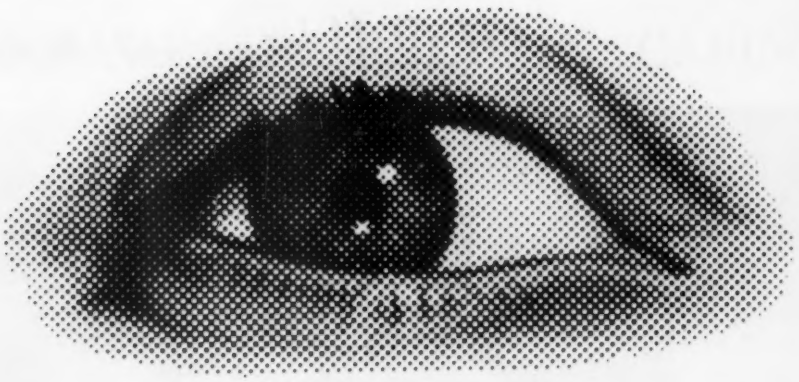

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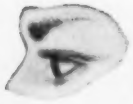

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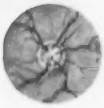

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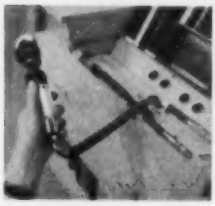

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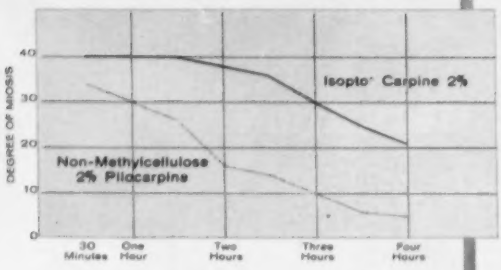

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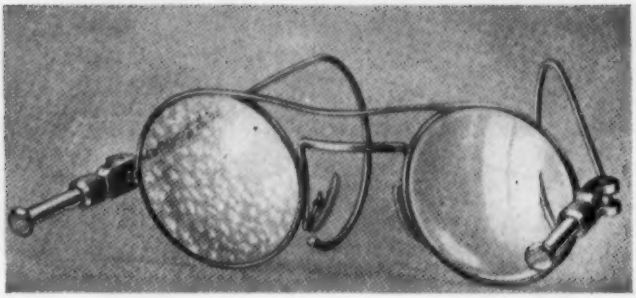

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Nevoxanthoendothelioma with Ocular Involvement

A Report of Two Cases

FRANK W. NEWELL, M.D., Chicago

Nevoxanthoendothelioma is a comparatively uncommon benign dermatologic disease characterized by the appearance in infants of one or more discrete bright-orange nodules on the skin. No abnormality of serum lipid values can be demonstrated; physical examination, except for the skin tumors, is negative, and there is a tendency for slow spontaneous disappearance of the lesions. Visceral involvement is usually not observed. The skin lesions are usually observed at birth or shortly thereafter and vary in number from one or two to many hundreds. They vary in size from 3 to 10 mm. in diameter and are 3 to 4 mm. above the surrounding skin surface.

These cutaneous tumors disappear at the age of 3 or 4 years, leaving a slightly glazed flat scar. There is no residual systemic disease.

The disease has been described as nevoxanthoendothelioma only in the past 10 or 12 years. Prior to that, examples of the condition were listed as endothelioma, xanthoma tuberosum multiplex, and juvenile xanthoma. A number of case reports have appeared in the dermatologic literature, and it would seem that the disease is well known to dermatologists.

Involvement of tissues other than cutaneous occurs exceedingly rarely. Lamb and Lain,[1] in 1937, described the eruption of erythematous nodules in a child at the age of 3 months. At the age of 16 months, the liver was enlarged, the lungs were infiltrated, and the blood lipids were elevated. These abnormal signs regressed spontaneously, and at the age of 5 years the child appeared to be entirely healthy. The authors felt that nevoxanthoendothelioma may be the connecting link between Schüller-Christian disease and cutaneous xanthomas—a theory which has gained considerable favor since then.

Ellis,[2] in the discussion of the paper of Lamb and Lain, described a lesion of nevoxanthoendothelioma in the right eye of a year-old infant. Further details are lacking, but apparently the tumor disappeared spontaneously.*

In 1949 Blank, Eglick, and Beerman[3] described a 4-year-old Negro with a pigmented mass in the eye which led to enucleation. The ciliary body and iris were found to be infiltrated and replaced with invasive cells. The condition was diagnosed as nevoxanthoendothelioma. Three weeks after the enucleation typical skin changes were noted. This apparently is the first case report in which a histologic diagnosis of involvement of tissues other than the skin had been made.

Theodore Sanders, at the meeting of the Eye Pathology Club in 1954, and Parker

From the Department of Surgery (Ophthalmology), The University of Chicago.

* Since this paper was submitted for publication Dr. Ellis has advised that the tumor involved the eyelid and that the globe was not affected.

Heath and A. Edward Maumenee, at the meeting in 1955, each presented examples of ocular involvement in nevoxanthoendothelioma. Maumenee [4] presented his findings at the 1955 meeting of the American Academy of Ophthalmology and Otolaryngology, but the other cases have not been published. Thus, there are three reports of ocular involvement occurring in the disease in the medical literature.

The disease was first described by McDonagh,[5] a pathologist, in 1909 and again in 1912. Nomland [6] has pointed out that the term nevoxanthoendothelioma was based upon several assumptions with which it is doubtful that anyone would now agree. McDonagh interpreted the histologic findings as being those of endotheliomata, and he looked upon the lesions as nevi, since they were present at birth in one of his patients. He regarded the xanthomatous process as one of dissolution of the initial disease. It is generally conceded at present that the disease is characterized by histiocytosis and that lipid infiltration of the cells is a secondary phenomenon.

However, even though nevoxanthoendothelioma is not an accurate term, it is well recognized and, according to Lamb and Lain,[1] it is fairly descriptive of a congenital malformation in which one encounters peculiar endothelial giant cells, as well as xanthoma cells, histiocytes, and fibroblasts, and, clinically, spontaneous involution of the lesions in later childhood.

The nature of the lesion in nevoxanthoendothelioma has occasioned considerable comment, particularly concerning its relationship to other disturbances of the reticuloendothelial system. Nomland [6] states that it must be considered as one of the xanthomatous diseases because of the presence of a typical foam cell in early lesions. The endotheliomatous appearance of the older lesions would then be considered a tissue reaction to the presence of cells containing fat, and their origin would be from histiocytes.

Farber,[7] in a series of observations, grouped together eosinophic granuloma of the bone, Hand-Schüller-Christian disease, and Letterer-Siwe disease. He concluded that "1. These are all variations of the same disease, with the clinical picture dependent upon the sites involved and the rapidity of the course. 2. Lipid, when present, is liberated as part of the local destructive inflammatory process. 3. There is no evidence that these diseases represent errors of lipid metabolism. There is no justification, therefore, for classifying these diseases with Gaucher's disease and Niemann-Pick disease. 4. A satisfactory working classification of these conditions should include the three separate clinical types of Hand-Schüller-Christian disease, Letterer-Siwe disease and eosinophilic granuloma of bone, with transitional forms between eosinophilic granuloma and each of the other two diseases of this group. 5. These conditions are probably of infectious origin."

In turn, Crocker [8] clarified the relationship of these conditions to each other with the following classification of cutaneous xanthomas:

Type I. Serum cholesterol (and fats) normal
- Group A. Xanthoma disseminatum (including those originally called "nevoxanthoendothelioma" and juvenile xanthoma)
- Group B. Xanthomas accompanying various chronic diseases, especially the "eosinophic granuloma syndromes" or the so-called "reticuloendothelioses" (including Hand-Schüller-Christian disease and Letterer-Siwe disease)

Type II. Serum cholesterol (and fats) elevated
- Group A. Xanthoma tuberosum (including essential familial hypercholesterolemia)
- Group B. Secondary or eruptive xanthomas (including the transitory xanthomas of diabetes mellitus, idiopathic familial hyperlipemia, nephrotic syndrome, hypothyroidism, obstructive liver disease, and von Gierke's disease)

Lichtenstein [9] has emphasized the role of the histiocyte in eosinophilic granuloma of the bone, Hand-Schüller-Christian disease, and Letterer-Siwe disease and suggested the name "histiocytosis X" for specific reference to the disease. He believed this term to be appropriate because of the connotation of inflammatory proliferative reaction as the common feature of all of the various pathologic expressions of the disease, whe-

Histiocytosis X *

Distribution of Lesions		Clinical Expressions	Age Incidence	Treatment	Prognosis
Localized in bone (one, several, or many foci); no discernible visceral involvement		Eosinophilic granuloma of bone	Infants, children, and young adults (occasionally older adults)	Curettement or x-ray therapy	Cure (although additional skeletal lesions may sometimes appear)
Disseminated	Acute or subacute course	Letterer-Siwe syndrome	Infants and young children below age of 3 years; occasional young adults (adult counterpart of L-S syndrome)	As yet nonspecific: Supportive—Antibiotics for secondary infections; x-ray therapy for skeletal and cutaneous lesions	Serious, though probably not invariably fatal; in occasional children, disease may become chronic or go into remission
	Subchronic or chronic course	Schüller-Christian syndrome (not necessarily Christian triad)	Children and young adults; occasional older adults	As for L-S syndrome; also x-ray therapy or β-hypophamine for diabetes insipidus; x-ray therapy for early pulmonary infiltration; cortisone?	Guarded, especially for children showing active progression and for patients (adults included) with pulmonary fibrosis and/or pituitary involvement

* Reproduced from Lichtenstein,[6] *A. M. A. Archives of Pathology*, American Medical Association, Chicago.

ther or not these lesions present also a concomitant conspicuous content of eosinophils and whether or not they show secondarily significant uptake of lipid by histiocytes. The clinical characteristics of these conditions are shown in the Table. Maumenee [4] related the skin and ocular changes in the patient described by him to the granulomatous phase of eosinophilic granuloma.

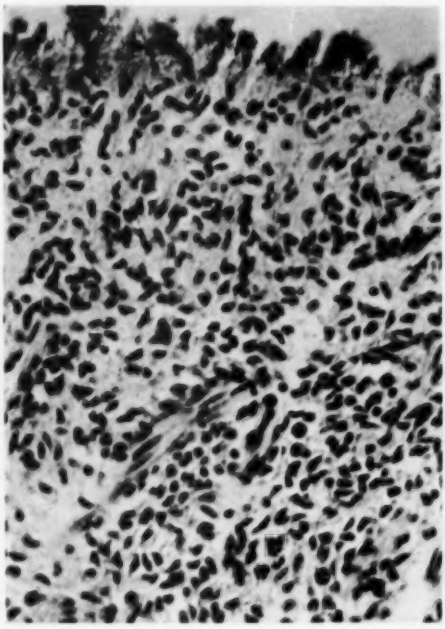
Dermatologists are nearly unanimous in classing nevoxanthoendothelioma as a benign cutaneous disease with onset in infancy characteristically without residue in several months or years. It is evident, therefore, that the occurrence of cutaneous and ocular signs together tends to give nosologic identity with systemic histiocytic conditions. Thus it seems likely that conditions of eosinophic granuloma of the eye or ocular involvement in Hand-Schüller-Christian disease may be forthcoming. Additionally, the ocular involvement, combined with cutaneous disease designated as nevoxanthoendothelioma, may be a connective link between this disease and Letterer-Siwe disease, eosinophilic granuloma, and Hand-Schüller-Christian disease.

Report of Cases

CASE 1.—A white girl was first seen at the University of Chicago Clinics when she was 10 months of age. The mother stated that when the infant was 4 months old it was noted that the left eye was extremely sensitive to light and that the cornea was cloudy. A diagnosis of glaucoma was made elsewhere at this time, and a miotic was prescribed.

Physical examination revealed a pale but apparently healthy, well-developed, and well-

Fig. 1 (Case 1).—Dense infiltration of the iris with deeply staining cells resembling endothelial cells. Hematoxylin and eosin; original magnification × 540.



nourished female infant. There were dozens of small yellowish-orange plaques on the head, back, and chest. They varied in size from 3 to 6 mm. and varied from being entirely flat to being scarcely elevated. The cutaneous lesion was most inconspicuous. It had been noted when the child was 3 weeks of age by the family physician and had been attributed to a drug reaction. The remainder of the examination was noncontributory except for the ocular abnormality.

The hemoglobin was 7.2 gm.; the red blood cell count, 3,150,000, and the leukocyte count, 11,200. Differential study indicated 65% polymorphonuclear leucocytes, 30% lymphyocytes, 3% monocytes, and 2% eosinophils. This iron-deficiency type of anemia has been constantly present. Urinalysis showed no abnormality. Total lipids were 808 mg. per 100 cc.; total cholesterol was 212 mg. per 100 cc., and phospholipids were 230 mg. per 100 cc.

The right eye was entirely normal, and the disease was limited to the left eye. The cornea was steamy and measured 12 mm. in diameter, compared with 11 mm. on the opposite side. The pupillary reaction to light was normal. Intraocular pressure was 18.6 mm. Hg (Schiøtz) in the right eye and 36.8 mm. Hg in the left eye. The fundi were normal. Gonioscopy indicated the angle to be open but shallow; a nearly transparent lace-like tissue very slightly obscured details of the trabecular area. The iris on the left was of a darker color than that of the right eye.

A goniotomy did not reduce the intraocular pressure, and one month later a cyclodialysis was also ineffective. Five weeks after this procedure there was subconjunctival infiltration of fatty material in the inferior temporal quadrant, in the area where the sclera had been grasped with fixation forceps. A trephine operation was performed in the 12 o'clock meridan and was combined with a large peripheral iridectomy. Since this procedure the tension has ranged between 17 and 20 mm. Hg. How-

Fig. 2 (Case 1).—A giant cell in the iris stroma. Hematoxylin and eosin; original magnification × 1600.

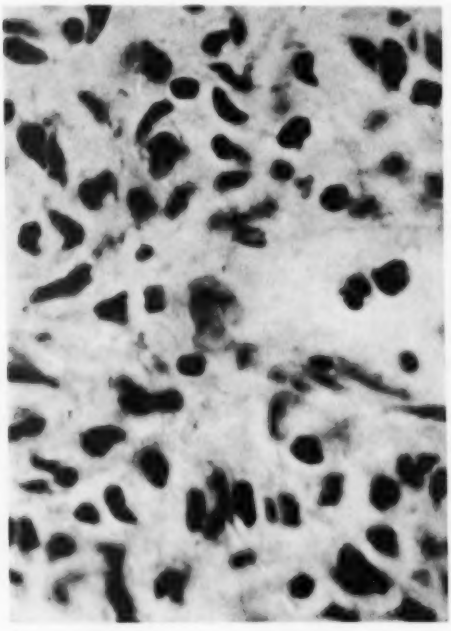
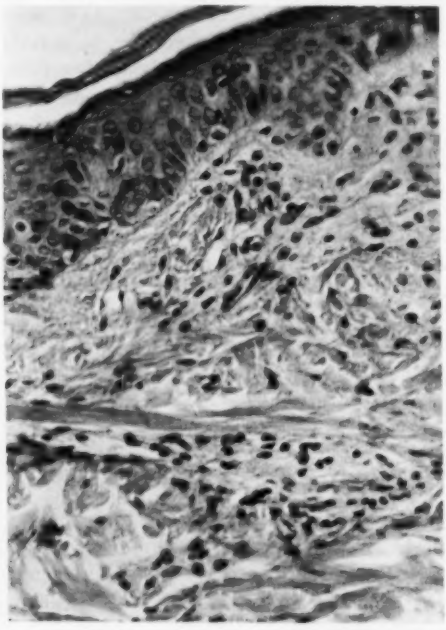


Fig. 3 (Case 1).—Section of an involved area of the skin. The infiltrating cells have same characteristics as the cells of the iris. Hematoxylin and eosin; original magnification × 470.



ever, the eye is divergent and vision is reduced to 2/300.

Microscopically the epidermis showed no abnormality. Throughout the corium there were nests of large histiocytic cells which had a pale-staining granular cytoplasm. In a few instances these cells seemed to be fused into the formation of large giant cells. The infiltrate extended from the deepest corium to the epidermis.

Microscopic examination of the iris and conjunctivia removed at surgery indicated a similar pathologic change in each. The iris was markedly thickened and generally infiltrated with histiocytes. Some of these histiocytes were fused, giving the appearance of giant cells. The blood vessels were dilated. There was no fatty infiltration.

Comment.—The diagnosis of nevoxanthoendothelioma by the dermatologists led to the recognition of the probable association between the skin disease and the glaucoma. The eye did not have a spontaneous hyphema, as occurred in the second case to be reported and which, according to Maumenee,[4] constitutes a characteristic feature of the disease. The normal blood lipids combined with the pathologic picture of histiocytic infiltration indicates the condition to belong to the group of histiocytosis of unknown cause.

CASE 2.—The patient was first seen at the University of Chicago Clinics, Jan. 8, 1938, when she was 2½ months old. The mother stated that, after an uneventful pregnancy and birth, the child had been entirely well until three days previously. At that time, the left eyelids were swollen and the eye was noted to be red.

The examination indicated the right eye to be entirely normal; the left upper and lower lids were moderately edematous, and there was ciliary injection. The cornea was partially opaque, and the lower portion of the anterior chamber was filled with blood. A number of physical examinations by pediatricians revealed no other abnormalities, and, in particular, there is no mention of any skin abnormality. The pupil was 2 mm. in size. The iris markings were very indistinct, and an exudate, gelatinous and plastic-like, was present. There was no red reflex. Tactile tension was normal. A diagnosis of metastatic uveitis with spontaneous anterior chamber hemorrhage was made. The patient was treated with local medications and two intramuscular injections of milk. This was followed by a decrease in the amount of exudate present in the chamber. Paracentesis was done on Jan. 18, 1938, by Dr. Louis Bothman, and examinations of aqueous humor removed showed many tumor cells which were interpreted as resembling retinal nuclear layer cells.

Fig. 4 (Case 2).—A section near the angle, indicating the massive infiltration of the sclera and iris. Hematoxylin and eosin; original magnification × 65.

On Jan. 2, 1938, a left enucleation was done with use of general anesthesia. A diagnosis of melanosarcoma of the ciliary body was made, and the patient received local radiation treatment to the orbit postoperatively. She was seen as a "feeding problem" subsequently in the pediatrics de-

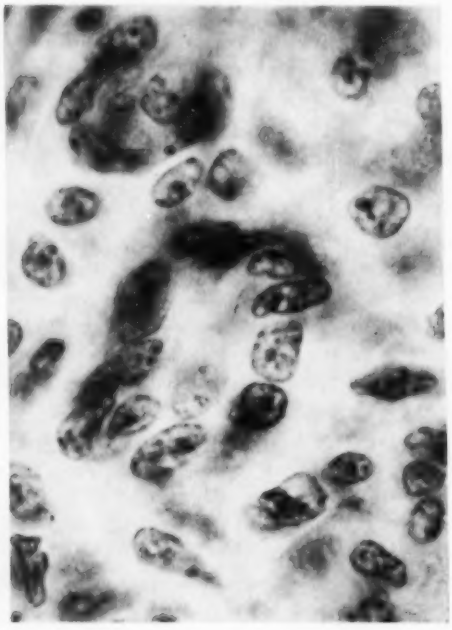
partment, and no abnormality was noted. She was last seen in the outpatient department in 1953, at which time the vision was 20/20 in the remaining eye, which was entirely normal. The anophthalmic orbit was normal, and the skin showed no disease.

The enucleated eye was fixed in Held's solution and sectioned horizontally. Serial sections 13μ to 17μ thick were made, and every 10th section was stained with hematoxylin and eosin.

The cornea perforated during surgery, and the iris prolapsed. The globe measured 18×11×18 mm. The cornea measured 11×9 mm. The sclera was thickened, particularly inferiorly, where it was overlying a tumor area. The tumor occupied the lower half of the anterior chamber and involved the iris and ciliary body. The remainder of the anterior chamber was filled with a tannish exudate.

Microscopic section indicated the optic nerve, choroid, and retina to be essentially normal, and the disease was limited to the anterior segment of the eye. The iris, ciliary body, and trabecula were almost entirely replaced by a dense infiltration of cells of varying size and shape. These cells had infiltrated the sclera and occluded the canal of Schlemm. A number of Touton cells were present, and there were many giant cells in the tumor area. An exudate containing erythrocytes and leukocytes, chiefly polymorphonuclear leukocytes, was present in the anterior chamber. An abscess of the cornea surrounded the area of prolapsed iris.

Fig. 5 (Case 2).—A large Touton cell in the stroma of the ciliary body. Hematoxylin and eosin: original magnification × 2220.



The diagnosis of the tumor as nevoxanthoma-endothelioma was not made until 1955, when Dr. Georgiana Theobold presented the eye to the Eye Pathology Club for discussion. The tumor cells consisted largely of histiocytes, which markedly infiltrated the iris and sclera. There was no fatty storage evident in these cells.

Comment.—The spontaneous hemorrhage into the anterior chamber, as occurred in this infant, has characterized the majority of cases of ocular involvement. The massive infiltration of the iris and sclera, with involvement of the angle and sclera, indicates that there was no possibility of saving the eye. Yet it must be recognized that the condition is a self-limited one and that if it is possible to prevent loss of the eye during the acute stage of the disease, spontaneous remission may well occur at a later date.

Summary

Two patients aged 10 months and 2 months are described, in each of whom histologic study of the eye indicated a histiocytosis of nevoxanthoendothelioma. The infant aged 10 months had typical cutaneous lesions and secondary glaucoma which was controlled surgically. The other infant had a spontaneous hemorrhage into the anterior chamber, and the eye was enucleated. The disease is characterized by a massive histiocytosis and may be a connecting link between the benign cutaneous condition known as nevoxanthoendothelioma and the systemic

diseases of eosinophilic granuloma and Hand-Schüller-Christian disease.

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SAMUEL T. ADAMS, M.D., Montreal, Canada

Pars plana cysts are occasionally seen when the extreme periphery of the fundus is examined with the Schepens technique [1,2] of indirect ophthalmoscopy and scleral pressure.

The cysts in this report were incidental findings in presumably normal eye bank eyes, examined before fixation by indirect ophthalmoscopy and scleral pressure and after fixation by histological study of the observed abnormalities.[3] The cysts were found in 1 of the 11 eyes studied, occupying the entire upper temporal quadrant (Fig. 1). The same eye had two round holes with opercula.

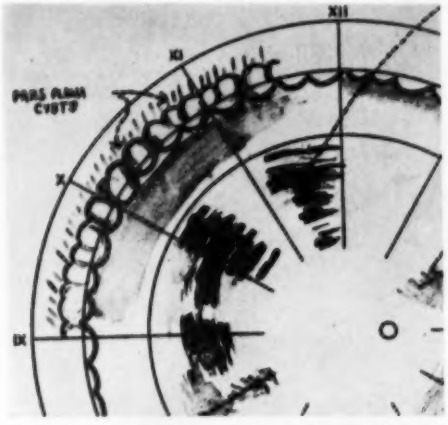


Fig. 1.—Retina diagram showing cysts throughout the entire upper temporal quadrant of the pars plana.

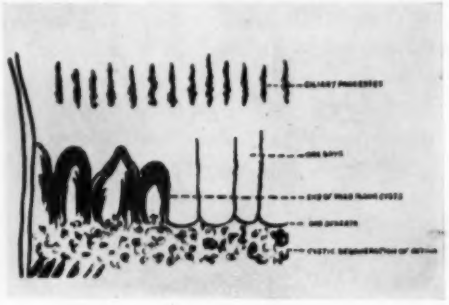
The cysts are oval and rather flat in shape. The lateral boundaries of each cyst are the individual ora bays. Posteriorly, they are continuous with the area of retinal cystic degeneration behind the ora. Anteriorly, they extend variably over about one-half of the pars plana toward the ciliary processes. The inner cyst walls are smooth and transparent (Figs. 2 and 3).

Fig. 2.—Photograph of pars plana cysts with ciliary processes anteriorly and vitreous fold over the ora area posteriorly.

Histopathology

(a) Meridional Section.—Toward the ciliary body there is a large empty cyst-like structure bounded on the scleral side by pigment epithelium and on the vitreous side by nonpigmented ciliary epithelium. More posteriorly there are several smaller cyst-like areas with elongated columns of nonpigmented ciliary epithelial cells be-

Fig. 3.—Diagram of junction of pars plana cysts area with normal pars plana.



From the Retina Service of the Massachusetts Eye and Ear Infirmary, Boston, and the Departments of Ophthalmology of the Montreal General Hospital and McGill University.

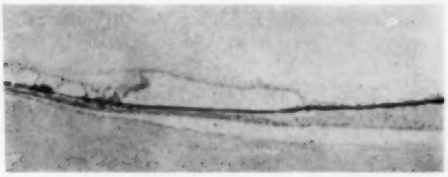


Fig. 4.—Pars plana cysts, meridional section, showing also anterior portion of cystically degenerated retina.

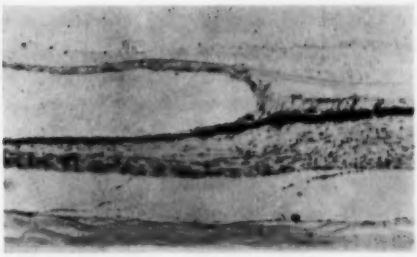


Fig. 5.—High-power view of pars plana cyst, meridional section, showing division of pigmented and nonpigmented epithelium.

tween. In some areas of attachment to the underlying structure, there are heaped up collections of pigment, while more posteriorly there is no pigment epithelium seen. Although the exact position of the ora cannot be determined, it must lie in this region, and the cyst-like structures posterior to the pigment epithelium probably correspond to the band of cystic degeneration in the retina.

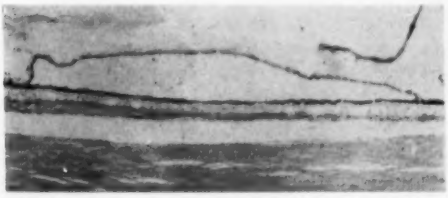


Fig. 6.—Pars plana cyst, transverse section.

(b) Transverse Section.—1. There are several large empty cyst-like spaces between the ciliary pigmented and nonpigmented epithelium. The areas of contact between the two types of epithelium usually show an increased amount of pigment, as though a proliferative process had occurred.

2. A section taken more posteriorly, presumably near the junction of the retina and ora, shows a cystic thinned retinal layer forming the inner wall of the cyst, with large cystic spaces separating it from the pigment epithelium.

Comment

Pars plana cysts have been observed ophthalmoscopically, as one of the forms of pars plana detachment.[1,11] Occasionally found at routine histological examination, they have been described along with cysts of the ciliary body and iris.[4,5] This coincidental appearance suggests that they accompany no specific ocular disease and have no clinical significance.[5] Their occasional observation clinically in both normal and many types of abnormal eyes supports this thought. It has been noted that some types of cysts of the anterior uveal tract may simulate tumors.[6,7] Multiple cysts of the ciliary body and iris have been noted gonioscopically.[8] One theory has been offered

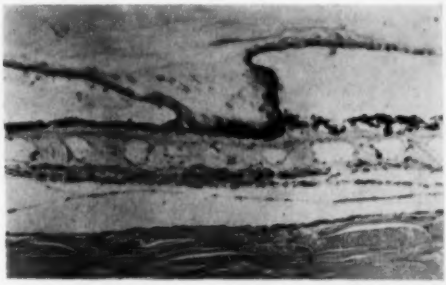
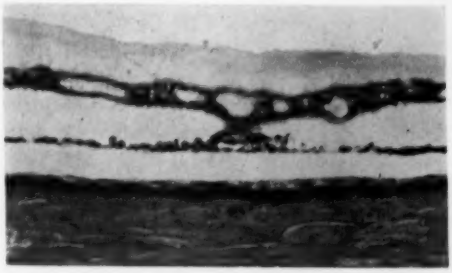


Fig. 7.—High-power view of pars plana cysts, transverse section, showing retina-choroid attachment between two cysts.

Fig. 8.—Pars plana cysts—a more posterior transverse section through area of cystically degenerated retina.



regarding the etiology of anterior uveal tract cysts.[9] Although their pathology has been described,[5] it has not been generally appreciated that they occur in the form and extent described here.

Summary

Pars plana cysts are occasionally seen by indirect ophthalmoscopy with scleral pressure. Single cysts have been reported as coincidental histological findings. This study correlates the ophthalmoscopic and the histological pictures. The cysts are apparently of no clinical significance.

The eye bank eyes were furnished by the Boston Eye Bank for Sight Restoration. Dr. David Donaldson photographed the pars plana cysts. Histological sections were made by the Department of Eye Pathology of the Massachusetts Eye and Ear Infirmary and photographed by the Photography Department of the Montreal General Hospital.

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Improvement in Hypertensive Retinopathy Following Adrenal Resection and Sympathectomy

Results in One Hundred Eleven Patients

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It is the principal purpose of this report to describe the retinal changes observed in 111 severely hypertensive patients after total or subtotal adrenalectomy combined with the Adson or the Smithwick type of sympathectomy.[1] Although improvement in the retinopathy of hypertension has been noted following various forms of medical and surgical therapy, the above-mentioned combined operations constitute a new approach to the treatment of essential hypertension and make available for study another group of patients. A secondary purpose of this report is to determine whether or not the ocular fundi can yield information that will be valuable in the selection of patients for whom such surgery can be beneficial.

Patients with severe hypertension show marked increase in peripheral vascular resistance and acceleration of arterial changes leading to impaired function and structural damage in such areas as the cerebrum, kidneys, heart, and retina. Moderate reduction in blood pressure can result in increased circulation, with a consequent reversal of some of these abnormalities.[2]

Such a reduction in blood pressure, according to Wolferth,[3] might best be obtained by attacking more than one of the mechanisms by which hypertension is maintained. This is a report on the results achieved by combining adrenalectomy with sympathectomy in certain carefully selected severely hypertensive patients who do not respond to intensive medical programs and who continue to show progressive vascular damage. The improvement in retinopathy noted during this study is at least a partial measure of the success of this method.

It has long been recognized that careful examination of the ocular fundus can aid in the evaluation of various forms of treatment for hypertension. While such examination is no substitute for complete clinical evaluation of the entire patient, accurate classification of the retinopathy may give some indication of the vascular integrity in certain other areas. There is evidence, for example, that functional and organic changes in retinal vessels may be correlated with those occurring in the kidney[4] and in the cerebrum.[5]

Extensive reports concerning the effects of a number of different forms of treatment have indicated striking improvement in the retinopathy of severely hypertensive patients (Table 1). The published results of combining sympathectomy with adrenalectomy are few. Cartier[11] reported that when sympathectomy was combined with adrenalectomy improvement of retinal lesions was noted in 90% of the patients, while after sympathectomy alone retinal improvement was noted in only 53% of patients. Revell[12] found that papilledema disappeared in all of three patients surviving the combined operation performed during the malignant phase of hypertension.

Method of Study

In March, 1950, a cautious experiment was undertaken by a group of surgeons and

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TABLE 1.—*Effects of Various Medical and Surgical Measures on the Retinopathy of Hypertension*

	Retinopathy	Patients, No.	Improved		Unchanged		Worse	
			No.	%	No.	%	No.	%
Rice Diet, (Newborg & Kempner[6]) *	Papilledema	100	95	95	5	5	0	
	Hemorrhages	90	85	94.0	5	6	0	
	Exudates	94	86	92	8	8	0	
Ganglionic blocking agents (Burnett & Evans[7]) †	Gr. III hypertensive	49	34	70	14	29	1	1
	Gr. IV hypertensive	22	17	77	5	23	0	
Sympathectomy (Smithwick[8]) ‡	Gr. I hypertensive	40	5	12.5	21	52.5	14	35
	Gr. II hypertensive	32	8	25	20	62.5	4	12.5
	Gr. III hypertensive	27	21	77.8	6	22.2	0	
	Gr. IV hypertensive	15	15	100	0		0	
Supradiaphragmatic splanchnicectomy	Angiospastic with or without hemorrhages	88	72	82	15	17	1	1
(Peet & Isberg[9]) §	Papilledema	17	17	100	0		0	
Splanchnicectomy (Huntington & Evans[10]) ∥	Gr. III hypertensive	64	54	84	9	15	1	1
	Gr. IV hypertensive	12	12	100	0		0	
Adrenalectomy & sympathectomy (Zintel at al.) ¶	Gr. II hypertensive	55	36	65.4	19	34.6	0	
	Gr. III hypertensive	37	33	89.2	4	10.8	0	
	Gr. IV hypertensive	18	18	100	0		0	

* Results after the rice diet are good in the severe grades of retinopathy. All hemorrhages and exudates had not disappeared, but with prolonged treatment, a higher percentage of improvement was noted.
† Patients were treated with Rauwolfia serpentina followed by pentoliniumtartrate (Ansolcysen). Only patients with Grade III to Grade IV retinopathy were included.
‡ Smithwick thoracolumbar sympathectomy. Note the higher incidence of improvement in the severer grades of retinopathy.
§ Patients were examined 5 to 11 years postoperatively. Patients with Grades I to III hypertensive retinopathy were all included in the same group.
∥ Patients were examined 4 to 12 years after splanchnicectomy.
¶ For complete summary of this series, see Table 3.

internists at the Hospital of the University of Pennsylvania with the object of determining the effectiveness of adrenalectomy in the treatment of hypertension.[1] After a small number of patients had been subjected to total or subtotal adrenalectomy alone, however, it was found that more effective control of arterial pressure and its symptoms could be obtained by combining adrenalectomy with a limited sympathectomy. The majority of the patients in this group, therefore, have had an Adson type of sympathectomy and either total or subtotal adrenalectomy.

All patients were subjected to a rigorous evaluation prior to operation. Specific etiologic factors, such as coarctation of the aorta, pheochromocytoma, polycystic disease of the kidneys, Cushing's syndrome, nephrolithiasis, glomerulonephritis, and unilateral or bilateral pyelonephritis, were, insofar as possible, excluded. The severity of the hypertension was tested by repeated postural blood pressure tests, and a clinical evaluation was made of the vascular damage in cerebral, renal, cardiac, and retinal areas. Repeated urinalyses, intravenous phenolsulfonphthalein excretion tests, urograms, electrocardiograms, orthodiagrams, and ocular fundus examinations were performed on all patients.[2] From the findings of these preoperative examinations, patients were classified according to the severity of their disease and their likely prognoses, Smithwick's method of classification being used.

Those patients who did not respond to intensive medical programs and who showed evidence of progressive vascular damage in the heart, kidneys, brain, or eyes were considered candidates for surgery. Usually they were not over 55 years of age and their kidney function had not been markedly impaired.

While examination of the ocular fundi was a part of the preoperative evaluation of 168 patients so treated, adequate information concerning the postoperative retinal findings is available currently in only 111 patients. Wherever possible, examinations were conducted at intervals of three to six months, beginning six months after operation, and no patient surviving less than six months is included in the study.

In order that the eye ground examination might be of maximum value in the total evaluation of the hypertensive patient, changes in the retinal vasculature that are the result of hypertension solely were con-

TABLE 2.—*Criteria for Grading Ophthalmoscopic Changes of Hypertension and Arteriolar Sclerosis (Scheie)*

Hypertension		Arteriolar Sclerosis	
Normal	None	Normal	None
Grade I	Earliest recognizable arteriolar narrowing	Grade 1	Earliest recognizable increase in light reflex and minimal arteriovenous compression
Grade II	Narrowing more pronounced, localized irregularities	Grade 2	More marked arteriovenous compression
Grade III	More extreme narrowing and irregularity hemorrhages and/or exudates	Grade 3	Copper-wire arteries and still more marked arteriovenous compression
Grade IV	Signs of Grade III and papilledema	Grade 4	Silver-wire arteries

sidered separately from those changes that are manifestations of arteriolar sclerosis. Because it clearly distinguishes between functional and organic changes, the method of classification advocated by Scheie[14] was adopted (Table 2). Arteriolar narrowing, arteriolar irregularity, hemorrhages, exudates, retinal edema, and papilledema are thought to be indications of the severity of hypertension and are grouped from normal to Grade IV, in order of their severity. Widening of the arteriolar reflex stripe, arteriovenous compression, and copper- and silver-wire attenuation of the arterioles, on the other hand, are manifestations of arteriolar sclerosis and therefore are graded separately, also from normal to grade IV. Thus, a person with mild hypertension might have almost no hypertensive changes and yet show marked arteriolosclerotic changes. The converse might be true in a patient with severe hypertension of recent onset.

Results

Since March, 1950, the beginning of the study, 168 patients have been operated upon, with utilization of various combinations of total and subtotal adrenalectomy and the Adson or the Smithwick type of sympathectomy. Of these patients, 155 had the combined operation, while an initial group of 13 patients were subjected to adrenalectomy alone. The retinal study includes 109 patients who had the combined operation and only 2 who had adrenalectomy alone. Only those patients who survived postoperatively for at least six months have been included.

Table 3 indicates the over-all results of operation on the hypertensive changes in this group of 111 patients. There is a much higher incidence of improvement in those patients who had an initial Grade III or Grade IV hypertensive retinopathy than in those who had an initial Grade II retinopathy. There appeared to be very little correlation between the preoperative prognostic classification (Smithwick) and the actual postoperative incidence of improvement in hypertensive retinopathy. Table 4 indicates a similar response in all Smithwick groups. Improvement in hypertensive retinopathy was not dependent upon an improvement in blood pressure. Table 5 reveals a corresponding incidence of improvement in hypertensive retinal changes among groups with and without improvement in blood pressure.

The relationships between the preoperative degree of arteriolar sclerosis and the postoperative blood pressure are tabulated

TABLE 3.—*Incidence of Improvement in Hypertensive Retinopathy Postoperatively* *

Preoperative Status		Postoperative Status Final Grade						
Original Group	Cases, No.	0	I	II	III	IV	Improvement, %	No Change, %
I	1	0	1	0	0	0	0	100
II	55	0	36	19	0	0	65.4	34.6
III	37	2	13	18	4	0	89.2	10.8
IV	18	0	2	14	2	0	100	0
Totals	111	2	52	51	6	0	78.4	21.6

* The highest incidence of improvement was found in the severe grades of retinopathy. See also Smithwick's series in Table 1. Patients have been followed for at least six months, and some, for as long as seven years.

TABLE 4.—*Postoperative Blood Pressure Responses in Relation to Preoperative Smithwick Group* *

Smithwick Group	Improved		No Change	
	No.	%	No.	%
II (39 pts.)	31	79.5	8	20.5
III (30 pts.)	21	70.0	9	30.0
IV (42 pts.)	35	83.3	7	16.7
Totals 111 pts.	87	78.4	24	21.6

* The Smithwick classification is an estimate of prognosis based upon an evaluation of preoperative clinical and laboratory findings. Grade IV represents the worst prognostic group.

in Table 6. There appears to be a slightly better prognosis in those patients with smaller degrees of arteriolar sclerosis, but the numbers are too small to be of statistical significance. There was no appreciable increase in the degree of arteriolar sclerosis after operation in the majority of patients

TABLE 5.—*Improvement in Hypertensive Retinopathy in 111 Patients With and Without Improvement in Blood Pressure* *

Patients, No.		Retinopathy			
		Improved		No Change	
		No.	%	No.	%
B. P. improved	91	71	78	20	22
B. P. not improved	20	16	80	4	20
Totals	111	87	78.4	24	21.6

* Improvement in hypertensive retinopathy was not dependent upon an improvement in blood pressure.

(Table 7). Progressive increase in the degree of arteriolar sclerosis was noted in a few patients, however—about equally divided between the group that had demonstrated an improvement in blood pressure and the group that had not.

TABLE 6.—*Effect of Retinal Arteriolar Sclerosis on Blood Pressure Response*

Preoperative Arteriolar Sclerosis		Postoperative Blood Pressure					
Grade	Patients, No.	Improved		No Change		Worse	
		No.	%	No.	%	No.	%
0	5	4	80	1	20	0	
1	40	35	90	5	10	0	
2	80	43	86	6	13.5	1	0.5
3	14	8	57.1	5	35.7	1	7.2
4	2	1	50	1	50	0	

* The prognosis for any individual patient cannot be predicted on the basis of the degree of arteriolar sclerosis existing preoperatively.

TABLE 7.—*Changes in Retinal Arteriolar Sclerosis in 111 Patients With and Without Improvement in Blood Pressure* *

Patients, No.		Arteriolar Sclerosis					
		Improved		No Change		Worse	
		No.	%	No.	%	No.	%
B. P. improved	91	9	9.8	62	68.1	19	22.1
B. P. not improved	20	1	0.5	13	65.0	6	30

* No significant improvement in arteriolar sclerosis was noted, and there was very little progressive increase in arteriolar sclerosis postoperatively.

Comment

Combined adrenalectomy and sympathectomy has proved to be an effective method of lowering the blood pressure in severely hypertensive patients, presumably because it attacks more than one of the mechanisms concerned in the maintenance of blood pressure levels. It may produce satisfactory results not to be expected after either adrenalectomy or sympathectomy alone. In successfully controlled patients convalescence has been briefer. Blood pressure relapses have not been observed after the combined operation. There has been a striking improvement in those patients having large hearts and a tendency toward pulmonary edema or congestive failure. As in other forms of surgical treatment for hypertension, patients subjected to this operation often exhibit marked clinical improvement in the face of poor blood pressure responses.

An interpretation of the changes occurring in the retinopathy of hypertension after combined sympathectomy and adrenalectomy must be based, at least in part, upon theoretical grounds. Many important questions concerned with the development and disappearance of retinal changes in hypertensive patients remain unanswered. Although hemorrhages, exudates, and edema are thought to be the result of disturbances in retinal blood flow, and therefore evidences of inadequate nutrition, experimental evidence concerning the validity of those deductions is lacking, since no technique has yet been devised for the measurement of retinal blood flow.

Certain studies of cerebral hemodynamics, however, may be of some aid in the interpretation of vascular changes observed in the retina. Since retinal and cerebral vessels have a common embryologic origin, a limited application to the retinal vessels of the results of cerebral vascular studies seems justifiable. Leopold and his co-workers [5] have demonstrated a statistically significant correlation between the grade of retinal hypertensive change and the cerebral vascular resistance. Kety and his co-workers [15] have demonstrated that cerebral oxygen consumption and cerebral blood flow remain normal but that cerebral vascular resistance is increased in patients with essential hypertension. In patients with hypertensive encephalopathy, however, Finnerty [16] thinks that there is a decreased cerebral blood flow. The effect of combined adrenalectomy and sympathectomy on cerebral vascular dynamics has been studied. Hafkenschiel, Friedland, and Zintel [17] found that cerebral vascular resistance was lowered toward the normal range and that cerebral blood flow and oxygen consumption were maintained at normal levels or were slightly increased following combined sympathectomy and adrenalectomy. Clark, Crosley, and Barker [18] found a similar diminution in over-all renal vascular resistance following combined adrenalectomy and sympathectomy. The mechanism by means of which vascular resistance is reduced following this operation and other antihypertensive measures is not known.

If these findings can be applied to the retinal vessels, then the improvement in the retinopathy of the patients reported in this study would seem to be in part the result of diminished vascular resistance. Operative relief of local areas of vascular resistance, regardless of the effect on over-all retinal blood flow, may thus be responsible for the improvement of exudates. Loss of sodium and water, on the other hand, occurs postoperatively in these patients and may contribute to the reduction of edema. Hemorrhages, exudates, and edema disappear very rapidly, while vascular narrowing and irregularity, probably because of the presence of arteriolar sclerosis, improve very slowly. As time passes, more patients are found to have shown improvement in these vascular changes. Although apparent narrowing and irregularity may persist in the larger vessels, owing to the presence of arteriolar sclerosis, the smaller peripheral branches may show considerable relaxation.

Since arteriolar sclerosis is an organic change in the wall of the arteriole, no improvement here could be expected following operation and control of blood pressure. It is of corroborative interest, therefore, that in this study we found no improvement in arteriolosclerotic retinopathy postoperatively, during a period of observation as long as seven years. Although there appears to be a slightly better prognosis for improvement of arterial blood pressure in those patients having smaller degrees of arteriolar sclerosis, it is apparently impossible to predict the results of operation in the individual patient on the basis of such changes.

Summary

An analysis of the preoperative and postoperative retinal findings in a group of 111 patients subjected to combined adrenal resection and the Adson or the Smithwick type of sympathectomy is reported.

Improvement in retinopathy was noted in 78.4% of this group of patients. Among those with Grade III hypertensive retinopathy, however, there was improvement in 92%, while all patients with Grade IV hypertensive retinopathy showed improvement. Hemorrhages, exudates, and edema disappeared rapidly, while arteriolar narrowing and irregularity improved more slowly.

Improvement in hypertensive retinopathy may appear without improvement in blood pressure following operation.

The degree of arteriolar sclerosis present preoperatively appeared to have little bearing on the final blood pressure response, and this finding could not be applied to the individual patient as a prognostic sign.

Improvement in retinopathy is probably the result of diminished peripheral resistance, with consequent improvement in retinal blood flow.

Drs. Irving H. Leopold and George Tyner performed many of the examinations during the early phases of this study. Drs. W. A. Jeffers, F. H. Adler, H. G. Scheie, F. C. Wood, and C. C. Wolferth gave assistance in the preparation of this manuscript.

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Malignant Melanoma of the Uvea

Observations on Growth and Behavior, Enucleation Refused or Delayed

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The Chairman's address before the Ophthalmic Section of the American Medical Association * testifies to the complexity of diagnosis in malignant melanoma of the uvea. Another aspect of this problem is the equally fascinating question of prognosis. When one is facing the decision of advising enucleation of a tumor-containing eye, where the other is missing or useless, or where the patient is aged or infirm, this matter of equating the degree of malignancy against life expectancy becomes a very real and pressing responsibility. It is by no means universally appreciated that these tumors are capable of a most variable and unpredictable behavior. The broad range of their activity extends from the clinically malignant but dormant tumors of unknown duration with a time fuse of unknown length, whose growth under observation is imperceptible, to the devastating malignancy which seems literally to explode with a shower of lethal metastases. These reports are submitted as a contribution toward the understanding of these peculiar growth characteristics. They comprise a group of presumed or verified malignant melanomas in which enucleation has been refused or was appreciably delayed. The opportunity for observation over a long interval has proven them to fall within the less malignant category when evaluated against the generally accepted range of activity for these tumors. These cases have been extracted from The Duke Hospital series of malignant melanomas followed 10 or more years. The mortality of this series of 56 cases parallels that of the much larger series of the A. F. I. P. The cases studied here are therefore considered as representative of average experience. If such an assumption is valid, it seems probable that a significant number of uveal melanomas fall within the range of low growth potential and that early enucleation is not always a mandatory life-saving measure. If in the future we learn to recognize and evaluate characteristics peculiar to these inert or slowly growing tumors, we may have generated one gleam of hope in an otherwise uncompromising picture.

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CASE 1.—In 1928, at the age of 40, a woman first noted blurring of vision. Eighteen months later a colleague (Dr. Frank Smith) diagnosed a malignant melanoma and advised enucleation. This diagnosis was questioned and enucleation was refused after another consultant made a diagnosis of chorioretinitis juxta papillaris. Eighteen months later, some three years after the first symptoms, this blind painful glaucomatous eye, containing a large spindle-cell melanoma, was enucleated (Fig. 1*A* and *B*. For 28 years this person was symptom-free. In November, 1955, headache developed. A dark nodule was discovered at the hair line over the occiput. Later numerous nodules appeared in the skin. Biopsy (Fig. 1*C* and *D*) confirmed the melanotic character of what was interpreted as a late metastatic lesion from the uveal melanoma. The similarity of the tumors is apparent in the microscopic sections (Fig. 1*B* and *D*). Metastases appeared rapidly in the lungs and liver. Death occurred in 1956.

CASE 2.—In 1946, at the age of 24, a Negro woman presented herself for refrac-

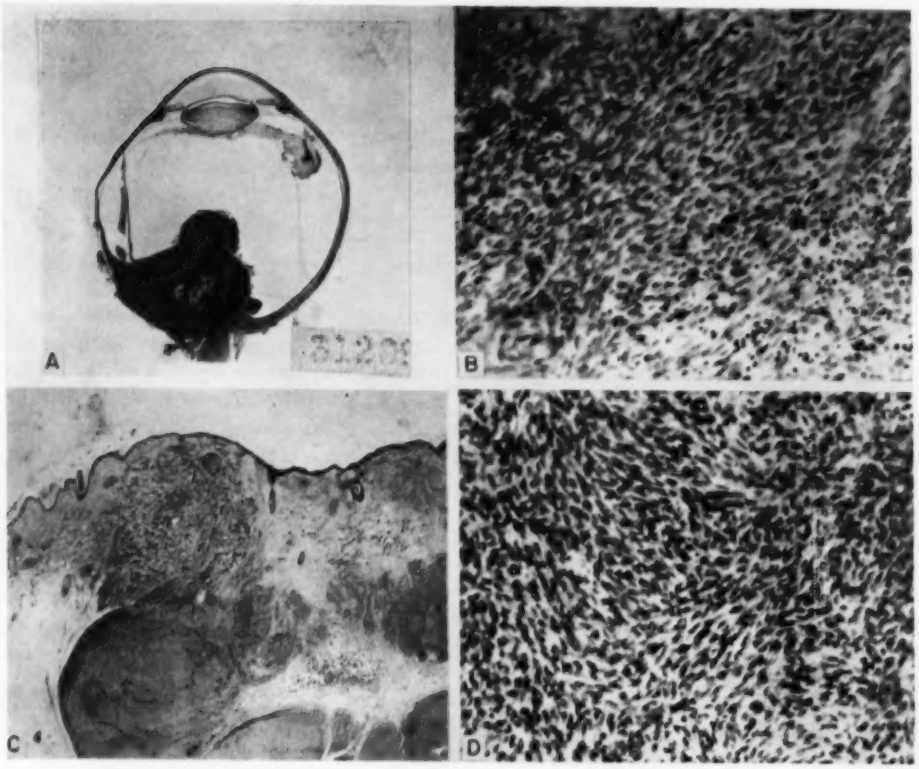

Figure 1

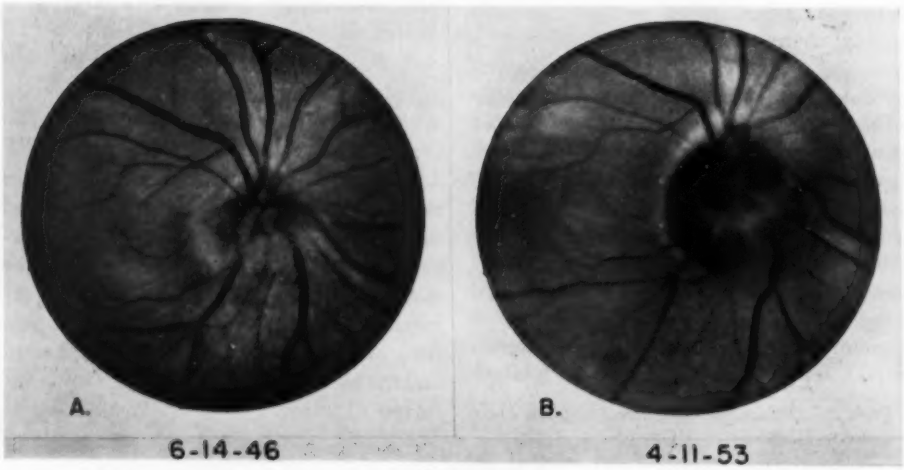

Figure 2

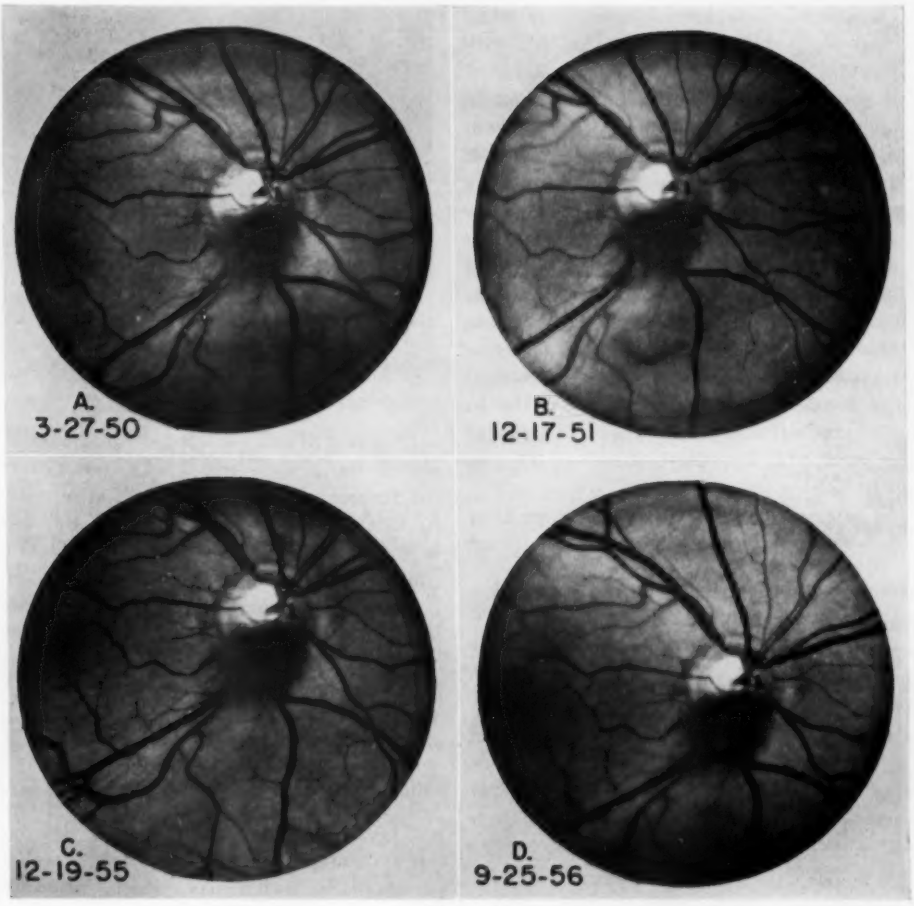

Figure 3

tion. Vision was 20/20 O. U. The right disc was found to be partially obscured by a circular domed mass of slightly greater diameter, superimposed half on the disc and half on the peripapillary retina below (Fig. 2*A*). The tumor had mounded up beneath the larger retinal vessels, displacing them forward. The upper border was sharply outlined against the white background of the disc. The lower border was less definite, plume-like strands feathering into the vitreous. The surface, as seen with the binocular ophthalmoscope and Hruby lens, resembled fine sleek, well-groomed fur. Enucleation was refused. In 1953 the tumor was larger and darker (Fig. 2*B*). Whereas in 1946 the upper third of the disc was visible, now only the barest crescent could be seen above the tumor. Uncorrected vision remained 20/30. Field studies were not permitted. This patient will not return for observation. She is reported to be in good health.

CASE 3.—In 1950 a white man, aged 42, during an allergy survey, was found to have the right disc partially obscured by a dark domed mass. The upper border of this mass, superimposed on the disc and of slightly larger diameter, was sharply defined. The lower border was indefinite.

Tendrils of dark brown tissue originating from the surface of the lower half proliferated into the vitreous (Fig. 3*A*). A diagnosis of malignant melanoma was made. Enucleation was refused. A year later vision remained 20/20. The blind spot showed a slight increase in the form of a projecting tongue corresponding to the distribution of the lesion. There was no demonstrable change in the size or shape of the lesion (Fig. 3*B*). In 1955 vision remained 20/20. There was questionable extension of the blind spot in a direction compatible with the long axis of the tumor. The tumor was unchanged (Fig. 3*C*). In 1956 vision remained unchanged. There was no perceptible increase in size or extension (Fig. 3*D*).

The third case shows striking simularity in form and location to our second case as it appeared in 1946. It has not, however, shown the growth which was observed in the former. Since enucleation was advised, in 1950, a lesion quite similar to this has been found in the literature, described as a congenital pigmentation of the disc.[1] Congenital pigmentation this may be; nevertheless, most ophthalmologists would probably be unhappy harboring such a lesion. This patient seems quite content with the status quo. His ophthalmologist, having advised enucleation six years previously, becomes increasingly uncomfortable.

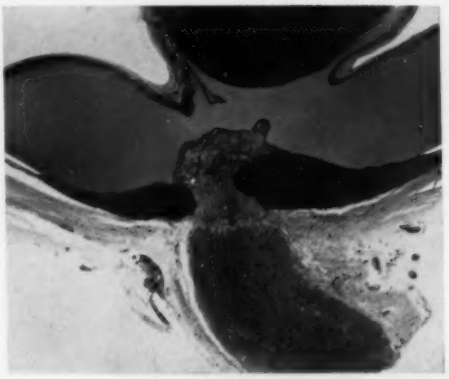

Figure 5

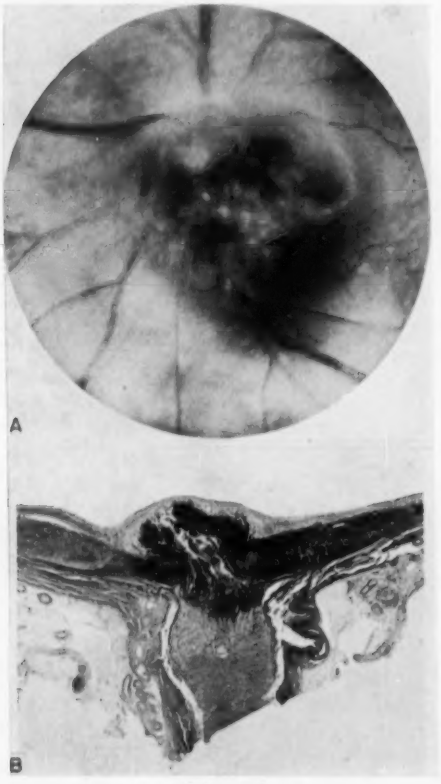

Figure 4

The following two cases are presented not because enucleation was long delayed but as examples of microscopically verified peripapillary melanomas which probably represent the same growth pattern as in the preceding cases.

Case 4.—In 1942 a Negro man, aged 32, presented with a history of failing vision of the right eye for the previous 12 months. Vision was 20/400. The disc was found to be masked by a chocolate-colored mass having a smooth domed surface. Streamers propagated into the vitreous (Fig. 4*A*). The eye was enucleated. The clinical diagnosis of a spindle-cell malignant melanoma was confirmed (Fig. 4*B*). The patient survives.

This tumor was unusual, first, in that malignant melanomas are rare in the Negro and, second, in location, having, as in Cases 2 and 3, originated presumably in the choroid adjacent to the disc and having

grown over and around the latter structure without extensive invasion of the nerve. This resistance of the papilla to invasion is more graphically shown in the following case.

CASE 5.—In 1953 a white man, aged 50, presented with a history of decreased vision in the left eye of four weeks' duration. A melanoma was found obscuring the disc (Fig. 5). If the history is correct and visual failure only developed four weeks before enucleation, the tumor had possibly grown very rapidly. More probably it had grown slowly over a long period without interfering with function. Here the tumor actually bridged the disc without interference in function of the nerve. The patient remains well.

CASE 6.—In 1937 a 26-year-old white man was seen with a history of gradually diminishing vision in the left eye for the preceding year. Vision was 20/200. A large smoothly domed tumor was seen obscuring the disc. Enucleation was at this time refused. One year later this eye was blind. The eye was enucleated. The tumor was found to be a spindle-cell melanoma. Unfortunately, the specimen has been lost and available sections are useless for finer differentiation. Drawings and photographs of the eye would indicate that this tumor, as those previously reported, arose from or near the disc margin (Fig. 6).

In this group of cases the tumors in each instance developed in close relationship to the disc. The first of these patients died 29 years after enucleation with late metastatic recurrence. The other four survive. In two of these the nature of the tumor has been confirmed by operation. In the two unoperated-on cases the fundus photographs show the marked similarity of morphologic pattern, although the growth rate is very different. The last case, with the large tumor neglected for a year, would on the basis of size alone have been expected to develop metastases. The behavior and growth pattern of these juxta papillary and peripapillary tumors suggests a more favorable prognosis than can be expected in the group as a whole.

In the preceding case reports we have described melanomas where the diagnosis appeared obvious but where enucleation was refused or deferred by patient's choice. Diagnosis is not always so easy. In certain patients the nature of the pigmented lesion can only be determined by observation over a period of months. The ophthalmologist must accept this calculated risk. A problem of this type is represented by Case 7.

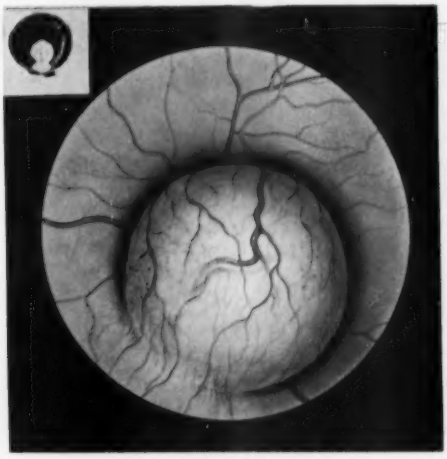

Figure 6

Figure 7

Case 7.—In 1953 a 24-year-old white woman, during the course of a routine physical examination, was found to have a discoloration of the upper temporal quadrant of the right disc. Vision was 20/20. Fields were within normal limits. Accumulation of pigment on the surface of the disc or within the cup is of course not uncommon, particularly after retinopexy or other chorioretinal inflammation. Indeed, one may occasionally mistake the shadow cast by the overhanging edge of a deep eccentric cup for pigment within the disc. In this case (Fig. 7, taken Dec. 11, 1953) the pigment proliferation appeared to have developed within the nerve fiber layer and not, as in the preceding cases, to have proliferated from the contiguous choroid. The upper temporal quadrant was slightly raised, rolled, and everted. Pigment had spread down into the cup and out into the retina. While the diagnosis was uncertain in this case, there has been no appreciable change in appearance and enucleation does not at this time seem indicated. This lesion may be a bona fide "congenital pigmentation." On the other hand, Cordes and Hogan have reported a malignant melanoma arising directly from the nerve.[2] Time may prove this to be another such case.

The preceding case has demonstrated the difficulty of diagnosis when a satisfactory view of the lesion is possible. The following case is an example of an early error in diagnosis due in part to poor visibility of the tumor and in part to other factors later proven to be extraneous.

Case 8.—In 1939 a 68-year-old physician complained of bilateral visual failure. He was known to have diabetes, arteriosclerosis, hypertension, and syphilis. His best corrected vision was O. D. 10/400, O. S. 20/40. The posterior segments in each eye were poorly visualized as a result of rather dense vitreous opacities, the right fundus being much more obscured than the left. Evidence of diabetic and hypertensive retinopathy could be indistinctly seen in the left fundus. In the lower temporal quadrant of the right eye the normal fundus reflex merged into a dark poorly defined shadow judged to be retinal separation associated with his constitutional disease complex. Two years later, in 1941, as a result either of vitreous clearing or of increasing prominence of the mass, the possibility of a neoplasm was considered. The eye was enucleated, and this impression was confirmed by the discovery of a melanoma (Fig. 8).

Figure 8

This man died in 1955 of a complication of diseases, 16 years after enucleation. Late enucleation does not seem to have decreased his life expectancy. In this instance the diagnostic picture was complicated by the associated diseases and by poor visibility. Case 8 exemplifies the fact that one should always seriously consider the possibility of tumor, even in the presence of other diseases, when a retinal separation is seen or suspected. We have recently seen an extensive recurrence of malignant melanoma in an orbit one year after enucleation of an eye which two years previously had been unsuccessfully operated on for retinal separation. In 1951 we were saved from a similar error. A middle-aged woman who had for six months following a bad fall complained of flashing lights in the left eye was found to have a bullous separation with two round holes and a linear tear. Opera-

tion was refused. Six months later she returned with a blind painful glaucomatous eye which was enucleated. Routine sections revealed a previously unsuspected melanoma. In the past 10 years three blind painful eyes enucleated at The Duke Hospital have been found on section to contain malignant melanomas. In these cases it was uncertain as to whether the tumor was responsible for the blindness and the subsequent inflammation or whether, a less likely possibility, a melanoma developed or was activated as a result of the irritative effect of the inflammatory reaction. In any case, the fact that such unsuspected melanomas are occasionally encountered presents a convincing argument for enucleation in preference to evisceration of blind chronically or recurrently inflamed eyes. The three cases cited here survive. It seems a reasonable assumption that the prognosis would not have been so favorable had these globes been eviscerated. In the general population there must be many such blind tumor-containing eyes. Only rarely does one hear of a metastatic lesion being traced back to such a previously undisclosed primary focus.

Even if the tumor is clearly visible, its true nature may not be recognized in the presence of other confusing signs. The following case illustrates this situation.

CASE 9.—In 1953 this white woman, aged 63, presented with a six-month history of failing vision of the right eye. She insisted that vision had greatly improved in the previous few weeks. Vision was 20/50. In the right eye superior and temporal to the macula was seen a pigmented, perhaps slightly elevated, mass of some 3 disc diameters in size. Superiorly and nasally, but definitely separated from this mass, was another lesion of somewhat similar character and size (Fig. 9). The possibility of malignant melanoma was considered but was at first discarded because of the improbability of two such tumors in the same eye. After 11 months of observation, during which time there was no apparent change in the shape or size of either tumor, enucleation

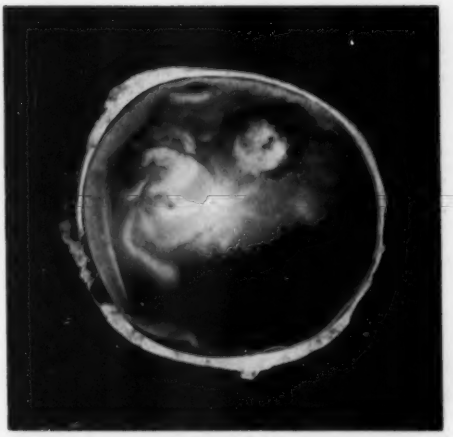

Figure 9

was advised. Two separate tumors, classified as spindle-cell A and B, respectively, were found. The patient remains well to date.

Prognosis in melanoma of the iris is said to be better than that of the choroid, presumably because it is accessible for early diagnosis. The following case indicates that other factors may be involved.

CASE 10.—In 1953 a lesion of the iris (Fig. 10) was observed in the course of a general physical examination of a 66-year-old woman. The patient stated that 24 years previously she had been advised that she had a melanoma in this eye. She presented a sketch drawn at that time, which in comparison with the tumor indicated no change in size. A somewhat cauliflower-like mound of grayish brown semitransparent tissue growing from the iris extended into the lower anterior chamber. At its base was a somewhat plaque-like irregular area of denser pigmentation. The angle was not involved. The iris proper was puckered around the borders of this basal plaque, which formed the foundation for the protruding portion of the tumor. The tumor was very vascularized. Clinically, the tumor was considered by three ophthalmologists to be malignant.

In this case the patient refused to consider a biopsy. In view of the long duration

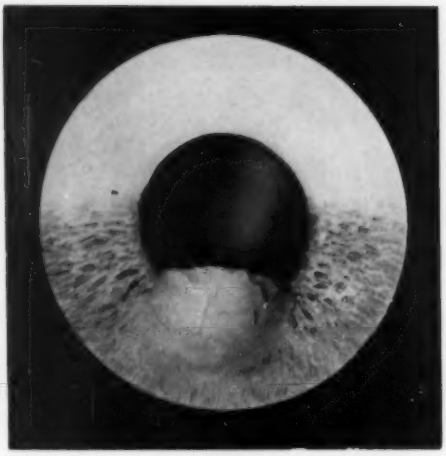

Fig. 10 (Case 10).—Melanoma, clinically malignant, dormant 20 years in a 69-year-old white woman. Picture taken March 1, 1956.

without increase in size, there was little basis for insisting on such an examination. Inactivity over a period of 25 years forces the conclusion that, regardless of appearance, this lesion is in reality a nevus of the iris. A study of the uptake of radioactive phosphorus (P^{32}) in lesions in this location may, with more experience in a larger group, correlated with known duration, as in this case, prove of great value in diagnosis.

It would seem appropriate to conclude this paper, as it was opened, with a brief consideration of late recurrences. In one instance we have seen the late development of a tumor in the fellow eye of one removed for malignant melanoma seven years previously.

Case 11.—In 1943, at the age of 51, this patient complained of gradual loss of vision for 18 months. Vision in the left eye was limited to form perception; corrected vision in the right eye was 20/60. There was extensive myelinization in the right fundus. The enucleated left eye contained a melanoma. In 1950 a melanoma was discovered in the right eye. Attempts to destroy this by electrocoagulation may or may not have been successful. Following this procedure the fundus was obscured by haze, and later, by extensive hemorrhage. A few months later the patient died with extensive metastases. Metastasis from one eye to the other has been reported. This may be such a case.

Late recurrences may develop in the orbit. They may appear as metastatic lesions in remote areas without local recurrence. They may reappear as local recurrences combined with metastatic foci.

Case 12.—Figure 11*A* is the photograph of a 48-year-old man who two years previously had the right eye enucleated because of a malignant melanoma. One year after enucleation pain developed in the right orbit. When he was seen in The Duke Clinic, a dark nodule had been present in the skin of the forehead for several weeks. The orbit was filled with tumor. The general condition was poor; the liver was enlarged. There was every indication of generalized metastases.

Case 13 (Fig. 11*B*).—In 1932 the left eye of a 49-year-old woman was enucleated. For two years previously the patient had

Figure 11

been troubled with "flashing lights." The eye was removed because of intense pain and inflammation. The patient was told that she had a tumor in the enucleated eye. A verified pathological report could not be obtained. In 1938 a hard growth appeared in the upper orbit. This was apparently excised, "drained and cut off." The patient first presented herself at The Duke Clinic in 1946 complaining of pain in the left orbit. A hard freely movable deep mass was felt in the orbit. An encapsulated roughly spherical rubbery coal-black mass, having a diameter of 2 cm., was found on exploration. The orbit was exenterated. In 1948, twelve years after enucleation and two years after exenteration, she died of the complications of direct intracranial extension without obvious signs of metastasis.

CASE 14 (Fig. 11*C*).—Another patient, in 1949, at the age of 64, had the left eye enucleated for a malignant melanoma. Six years later, in 1955, she returned because the prosthesis could no longer be worn. Local recurrence was obvious. The orbit was filled with a movable dense painless mass. The recurrence has apparently grown slowly without metastasis. The patient is still living, with relative comfort. She did not consent to evisceration.

Consideration of these last three cases brings up the question of orbital evisceration in late recurrence. In Case 12, because of obvious multiple metastasis, further surgery was obviously useless. In Case 13 the recurrent lesion was judged to be localized and was considered to have been completely removed in the eviscerated mass. Unfortunately, later developments indicated that the tumor had already spread beyond the orbital confines. It might be argued that surgical interference at this stage accelerated the growth and extension of the melanoma. The concepts of modern oncology would hardly support such a view.

Comment

Cases have been presented as illustrative of the low growth potential which may at times be observed in a class of tumor having a wide and variable range of malignancy. Microscopic classification of these tumors, with the implied correlation of degree of malignancy, has not been considered germane to the particular facet of the problem under discussion, i. e., the behavior of the lesion in vivo. Observation over a significant interval of these tumors supports the oncologic concept that, in addition to the physical restraint imposed by the sclera, lamina cribrosa, and Bruch's membrane, there must be other, biochemical, barriers, the understanding of which may in time lead to more discriminating, reliable, and conservative therapy than is at present available. Other classes of tumor undoubtedly exhibit individual differences in behavior and in ability to propagate and metastasize. The uveal melanoma is, however, peculiarly adapted for study because of the extreme range and variability of its growth potential and because it usually comes early under observation, it is easily accessible for observation, and it is usually "tagged" by its melanin production for identification when metastatic lesions develop.

Organs and even tissues of the body seem to possess an integrity of environment impervious to invading foreign cells. Thus it is that normal bowel cells do not metastasize to liver or skin to subcutaneous fat. Conversely, there is evidence that normal cells can duplicate themselves only in the presence of similar normal cells. It is said that single cells will not survive in tissue culture.

A neoplasm is by definition a new growth. It is noteworthy that all neoplasms are not really "new." In other words, the characteristics of the tissue of origin are usually easily recognizable in the early phases of its growth. And it would seem reasonable to suppose that this growth is possible because the neoplastic cell is sufficiently like its normal neighbor to respond to the same growth hormones. This kinship also protects the new cell from the normal tissue defenses responsible for the preservation of the "integrity of the environment." The requirements for the successful establish-

ment of a neoplasm would thus seem to be the following: 1. A cell different from its neighbors must arise and have the ability to reproduce itself. (One mechanism would be somatic mutation through external agents such as ionizing radiation. Another would be inherent instability of the genetic apparatus in "spontaneous" mutations.) 2. The new cell must be sufficiently responsive to local environmental conditions to reproduce itself at least as rapidly as its neighbors. For a neoplasm to become malignant, that is, invasive or metastatic, it must in addition have the following characteristics: 1. The neoplasm must produce this self-growth-stimulating substance in such amounts as to render it independent of the environment of its neighbors. 2. In order to become widely metastatic, this postulated self-growth-stimulating substance must be elaborated in such amounts and have such freedom of passage as to permeate the body tissues and create a favorable total body environment. In this case, the primary neoplasm may be considered as analagous to an endocrine tissue liberating a hormone which makes distant growth of its cells possible. The elaboration of this growth-producing substance by a carcinomatous prostate, as well as the indirect effect of castration, is a well-recognized instance of this relationship. Another such analogy is the effect of menstruation on an endometriotic lesion in the intestine. The melanomas we have described fit very satisfactorily into this concept. The pigmented neoplasm arises from pigmented tissue and presumably is at least in part responsive to the substances which influence the activity of the normal pigmented cells. Malignant melanomas, for example, are almost never seen before puberty. They are said to be stimulated by pregnancy. Certain of the melanomas we have described could be said to have no "endocrine" activity. That is, the neoplastic cells either are not present in sufficient numbers or are not biochemically active in such degree as to produce their own environmental "lake." In this developmental stage they are dependent on their non-neoplastic neighbors for stimulation and protection. However, the fact that a melanoma after discovery and under observation is stationary or slow-growing cannot be interpreted as a good omen. There is no security in such a situation. The biochemical or hormonal fluctuations which are inherent with aging, from puberty to senescence, may and probably do permit sporadic imbalances between the host defense mechanisms, that is to say the environment, and a cell which, through mutation, has become potentially malignant. Such an imbalance might also be produced by growth-stimulating factors produced by repeated or sustained irritation, as in the cases of the three blind glaucomatous eyes discovered on microscopic examination to harbor malignant melanoma. Such a cell once generated elaborates a substance which stimulates the growth of other tumor cells, which in turn produce more growth-stimulating substance. This feed-back mechanism once established may be capable of elaborating a growth-stimulation factor peculiar to itself. This substance spreading throughout the body creates a hormone reservoir wherein melanoma cells may grow at will. We can assume that in our first case, in which metastases without local recurrence developed 28 years after enucleation, a few tumor cells had already escaped the globe and had migrated into an area in which the environment was at least suitable for survival. From this focus, in later years, as a result either of a favorable shift in this environment or of a mutation into a more malignant phase of development, the lethal tumor erupted. It follows that complete removal of every neoplastic cell, even in a structure so readily accessible as the eye, is not always accomplished by simple enucleation. How else can one explain the late recurrence and metastases in Case 1, the remarkably slow growth in Case 2, the inactivity in Case 3? The remarkably slow growth with late recurrence and metastasis observed in the other cases reported can

likewise be rationalized by this theory. We do not intend to imply that tumor-containing eyes should not be enucleated as soon as the surgeon is committed to such a diagnosis. Indeed, this theory not only obligates the surgeon to the early removal of the primary lesion but implies that even the exenteration of an orbital recurrence or the excision of a readily accessible metastasis may prolong life. The behavior in these cases does seem to emphasize the fact that there are inherent restraints in the body and that in our search for control not only of melanomas of the eye but of malignancies in general the ultimate solution may not lie entirely in the realm of surgery but also in the bolstering of our natural defenses through the discovery and application of modalities which will stabilize and preserve the "integrity of environment."

Summary

Observations are reported on a series of stationary or slowly growing tumors of uvea. Instances are cited in which malignant melanomas have been found on microscopic examinations of eyes in which no tumor was suspected. Cases of late local recurrence and metastasis are described. A theoretical explanation for the pathogenesis of these lesions is offered. A study of these reports indicates that hope for future control of these tumors may lie in the understanding and regulation of the environmental factors which permit their growth and dissemination.

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Electrocardiographic Changes During Ocular Surgery and Their Prevention by Retrobulbar Injection

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In an attempt to investigate clinically some of the possible etiologic factors contributing to cardiac arrest during ocular surgery and to study potential means of their prevention, electrocardiography has been performed in the operating room on a series of patients undergoing various types of intraocular and extraocular operations under both local and general anesthesia.

Our interest in this problem was first aroused in 1955, when one of us (R. E. K.) experienced the death of a patient during surgery for retinal detachment wherein fatal cardiac arrest developed shortly after the preliminary manipulation of two extraocular muscles. A review of the literature concerning the potential relationship between ocular surgery and cardiac arrhythmias revealed many basic facts but also bared many areas of deficiency in our understanding of this relationship and, most important to us, very little evidence as to means of prevention of cardiac arrest in eye surgery. It is toward the determination of actual operating-room electrocardiographic changes provoked by ocular surgical maneuvers and toward the study of possible means of their prophylaxis that the evidence in this communication is directed chiefly.

The oculocardiac reflex has been well known since Aschner [1] and Dagnini [2] independently reported in 1908 upon the bradycardia produced by digital pressure applied to the eyeball. The physiology of this reflex was investigated thoroughly by Aschner, and the experimental and clinical literature has been reviewed by Van Dooren [3] and by Wagner.[4] The afferent arc of the reflex is known to be mediated by the ophthalmic division of the trigeminal nerve, while the efferent pathway is via the vagus nerve. Thus, whereas our general surgical colleagues are thoroughly familiar with the vagovagal reflex (wherein both parts of the reflex from parietal or visceral tissues are mediated by the vagus), we are here dealing with a trigeminovagal reflex.

Electrocardiographic studies of the oculocardiac reflex have been performed in normal subjects by Petzetakis,[5] by Daniélopolu,[6] by Sabena and Posteli,[7] and by Gillespie, Richter, and Wang,[8] who found bradycardia and, if too great a pressure were exerted, cardiac standstill. The last authors found that normal heart rate usually returned within 20 seconds after release of pressure. Clinically prolonged oculocardiac reflexes due to irritation of the orbital trigeminal endings by postenucleation hemorrhage have been reported by Bailey,[9] by Vialard,[10] and by de Treigny,[11] the last author observing that orbital injection of procaine and epinephrine served as effective treatment. Ocular trauma has also been invoked as a cause of prolonged oculocardiac reflex by Aubineau,[12] who produced it with subconjunctival injection of saline; by Mougeot and Duverger,[13] who reported it in five cases of World War I orbital injuries, and by Szokolik,[14] who observed it in three cases of retrobulbar hematoma. Störtebecker [15] described a prolonged oculocardiac reflex in a case of orbital and

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subconjunctival hematoma, obtained electrocardiographic evidence of the bradycardia, and discovered that pressure on the injured eye caused exaggeration of the bradycardia and the appearance of nodal rhythm which disappeared immediately on release of the pressure. Michaud [16] observed an oculocardiac reflex after an operation for retinal detachment. In experimental animals, Palm and Strömblad [17] found that the oculocardiac reflex could be elicited by blows on the eye, traction on the iris, and pressure on the retrobulbar tissues after evisceration. Finally, Sorenson and Gilmore [18] recently observed that traction on the medial or lateral rectus muscle in children or adolescents caused temporary cardiac arrest in one case and bradycardia or clinical arrhythmia as judged by physical signs in every case of a 17-case series of strabismus operations.

It is thus apparent that a variety of stimuli applied to and about the eye can initiate the trigeminovagus reflex and result in demonstrable cardiac changes. Furthermore, it is seen that previous work has pointed the way admirably for the present investigation. There evolved initially the task of investigating the cardiac effect of all possible ocular surgical maneuvers in the operating room, of extending these observations by serial electrocardiography during a variety of ocular operations, and then of investigating possible ways in which the observed trigeminovagal cardiac effects could be abolished.

Materials and Methods

The material for this communication consisted of 50 private cases involving 10 different types of extraocular and intraocular surgical operations studied in the operating room during the performance of the actual surgery. The types included strabismus surgery, enucleation, retinopexy and scleral buckling operations for retinal detachment, cataract extraction, fistulizing operations for open-angle glaucoma, cyclodialysis for aphakic glaucoma, peripheral iridectomy for narrow-angle glaucoma, blepharoplasty, conjunctivoplasty, and tear-sac surgery. Various stimuli were applied to and about the eye during the operations and serial electrocardiography was performed to determine and record the cardiac changes produced. The operations were performed under both general and local anesthesia. In the general anesthesia group, premedication with a belladonna-like drug was routine expect in two cases detailed below, several different anesthetic agents were employed, and an attempt was made to correlate the depth of anesthesia with the cardiac changes produced by ocular stimulation. The age range of the patients was from 8 months to 92 years. The ocular stimuli studied included digital pressure upon the globe (as used historically to initiate the oculocardiac reflex and as now used in conjunction with the retrobulbar injection in intraocular surgery); traction on, as well as stretching, suturing, clamping, and transection of, the extraocular muscles; traction on the eyeball during enucleation; transection of the optic nerve; direct pressure in the orbital apex after enucleation; incision into the globe; traction on and incision into the iris; grasping of the anterior lens capsule; intracapsular tumbling of the lens; suturing of ocular incisions; forceps stimulation of bulbar conjunctiva and of skin of the lids and brow; subconjunctival injection of saline or penicillin; the mechanical stimulus of the retrobulbar injection itself; surgical diathermy applied to the sclera, and all other eye-stimulating surgical maneuvers utilized during the commonly performed ocular operations. In the muscle-surgery group, observations were made during operations on the medial, lateral, superior, and inferior rectus muscles and on the inferior and superior oblique muscles.

When a given stimulus was found to produce a definite electrocardiographic change the stimulus was released, and upon return of the tracing to normal the stimulus was reapplied. If it was found to be re-

producible, a retrobulbar injection of an anesthetic solution was performed and the cycle was repeated. In this way, evidence was obtained concerning the possible prevention in man of the cardiac changes induced by the trigeminovagal reflex.

Results

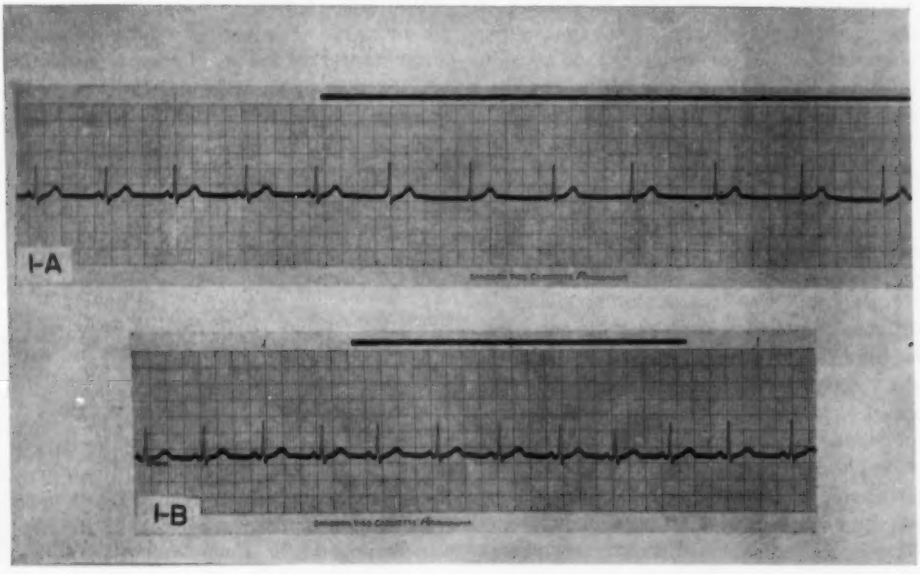
The only stimuli found to produce significant electrocardiographic changes were digital pressure upon the globe, manipulation of the extraocular muscles, and direct pressure upon the tissue remaining in the orbital apex after enucleation. These changes occurred under either local or general anesthesia, and under general anesthesia appeared as commonly under lighter (Plane I) as under deeper (bottom of Plane III) surgical anesthesia. There was no significant difference attributable to the choice of general anesthetic agent.

In the 50 cases studied, significant changes occurred in 15 patients, or 30%, while in the remaining 35 cases (70%) no significant electrocardiographic change was elicited by any ocular stimulus. In the 25 cases of extraocular muscle surgery, cardiac changes of consequence appeared on manipulation of the muscles in only 12 cases (48%), with no cardiac effect appearing in the other 13 cases (52%). This finding is at variance with the clinical (nonelectrocardiographic) observations of Sorenson and Gilmore,[18] who described bradycardia or ventricular fibrillation in every case of their 17-case series. A possible explanation for this difference will be discussed below.

As to the electrocardiographic changes considered significant, the commonest was the development of nodal rhythm (i. e., the appearance of a new cardiac pacemaker following suppression of the sinoauricular node); next commonest was marked bradycardia, and in two cases temporary cardiac arrest was produced.

Figure 1 represents the findings in a case typical of those showing significant electrocardiographic changes during horizontal muscle surgery. The patient was an 8-year-old girl undergoing a recession-resection operation for esotropia, with use of general anesthesia. Figure 1*A* illustrates the control Lead II in the initial portion of the tracing,

Fig. 1.—Effect of stretching medial rectus muscle.



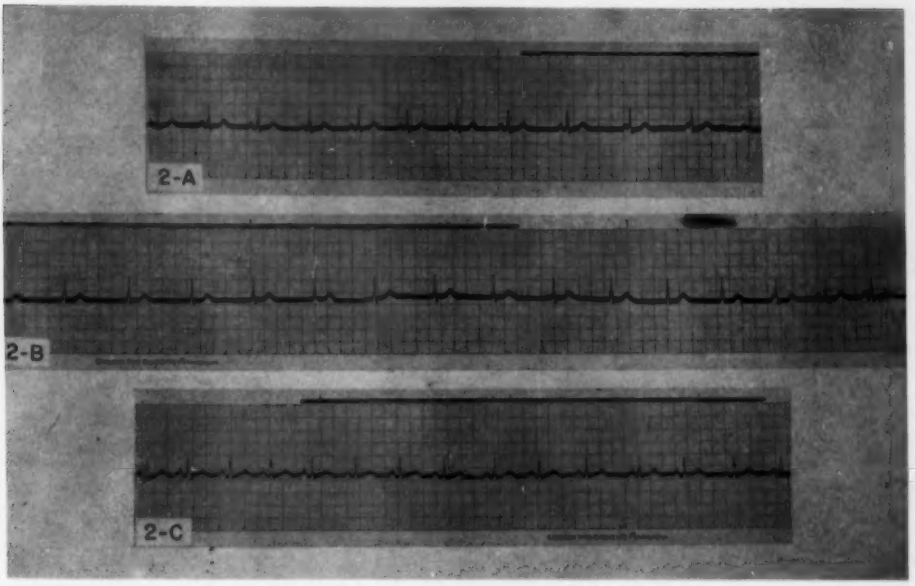


Fig. 2.—Effect of stretching superior rectus muscle.

wherein an upright P wave precedes each QRS complex. At the black marker, stretching of the medial rectus muscle was begun, and one sees the rapid development of bradycardia and nodal rhythm. Figure 1*B* demonstrates that the same stimulus, applied 90 seconds after retrobulbar injection of lidocaine (Xylocaine), provoked no electrocardiographic change.

Figure 2 depicts the changes in a 12-year-old boy undergoing surgery on a vertical muscle, with use of general anesthesia. Figure 2*A* portrays the development of bradycardia and nodal rhythm during the stretching of the superior rectus muscle. Figure 2*B* is a continuation of Figure 2*A*, and the return of sinus rhythm upon release of the muscle is readily seen. After retrobulbar injection (Fig. 2*C*) no electrocardiographic changes could be elicited by the same stimulus. In an attempt to determine which extraocular muscle, by its stimulation, could most easily provoke electrocardiographic changes, the infrequency of significant alterations in any one muscle did not allow a statistically valid conclusion. The highest incidence of positive changes occurred upon manipulation of the medial, lateral, and superior rectus muscles, while a significant change rarely occurred with stimulation of the inferior oblique and inferior rectus muscles.

It is of interest to observe that of the 15 cases of cataract operation studied none showed significant electrocardiographic changes induced by any ocular stimulus, including the acts of application of digital pressure to the globe and of grasping the superior rectus muscle and placing the superior rectus bridle suture. Since all other reflex-stimulating ocular maneuvers in intraocular surgery are performed after the retrobulbar injection, they need not be further considered here.

Similar findings of significant bradycardia were observed in an operation for enucleation of the eyeball. During extreme anterior traction on the globe and during transection of the optic nerve no cardiac change was produced, whereas with individual muscle manipulation or with direct pressure on the tissues remaining in the orbital apex, as done in order to secure hemostasis (and as Aschner had done in

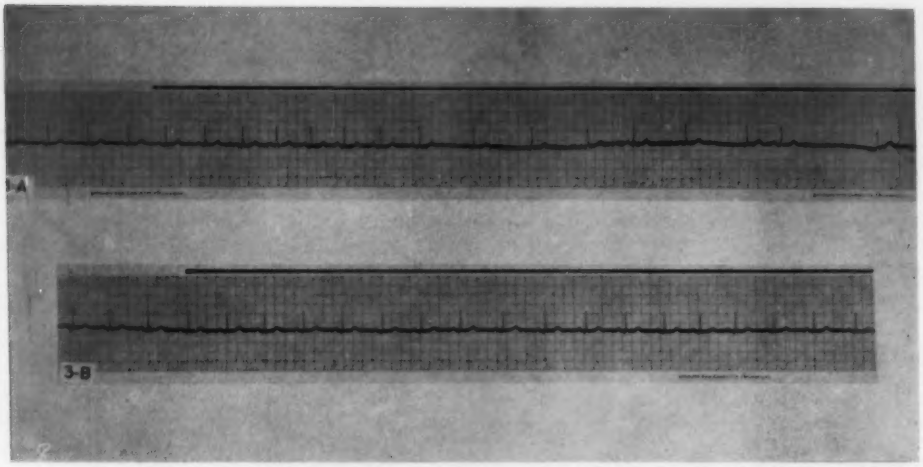


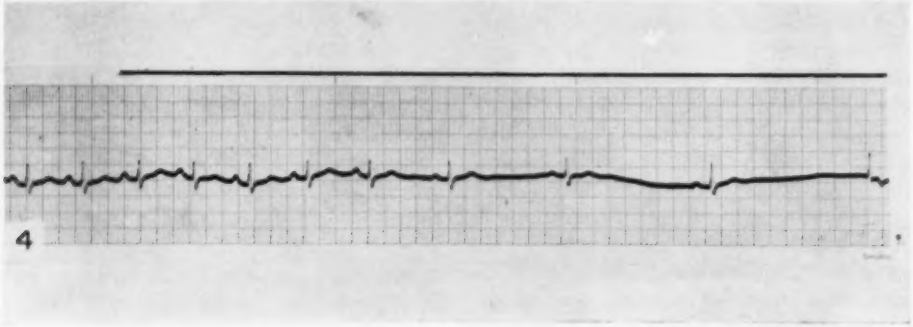
Fig. 3.—Transient cardiac arrest from digital pressure in case of excision of tumor of conjunctiva.

animals in 1908), significant bradycardia developed.

The two cases of transient cardiac arrest are represented in Figures 3 and 4. Figure 3 represents a 21-year-old woman being operated upon, with local anesthesia, for excision of a tumor of the bulbar conjunctiva, and the tracings depict the onset, with digital pressure to the globe, of bradycardia, nodal escape, and, finally, a transient cardiac arrest lasting more than two seconds. Following the return to normal, a retrobulbar injection was performed, and this abolished all further electrocardiographic changes (Fig. 3*B*). In Figure 4, representing the case of a 48-year-old woman undergoing surgery for correction of exotropia under general anesthesia, digital pressure applied to the eyeball produced similar effects and resulted in cardiac arrest lasting more than two seconds, during which the blood pressure decreased to zero. Again, retrobulbar injection completely abolished the reflex. The case in Figure 3 had had premedication with atropine, while the case in Figure 4 was one of the two cases in which general anesthesia was used where our anesthesiologist deemed it safe to omit an atropine-series drug, in order to eliminate any possible vagal blocking effect.

Of interest is the evidence presented in Figure 5, wherein the electrocardiographic effects of ocular stimulation are compared with the effects of vagovagal stimulation by

Fig. 4.—Transient cardiac arrest from digital pressure in case of exotropia.



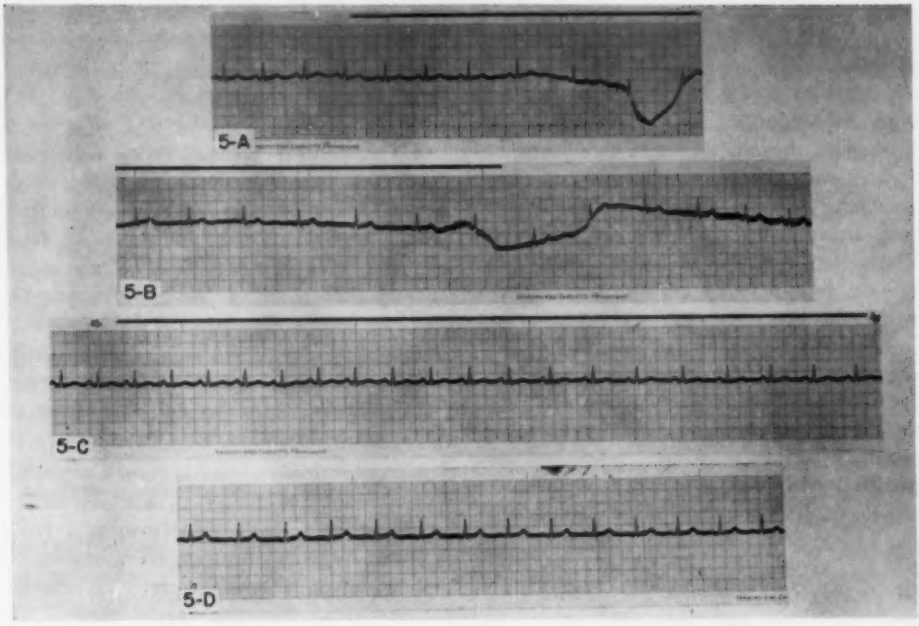
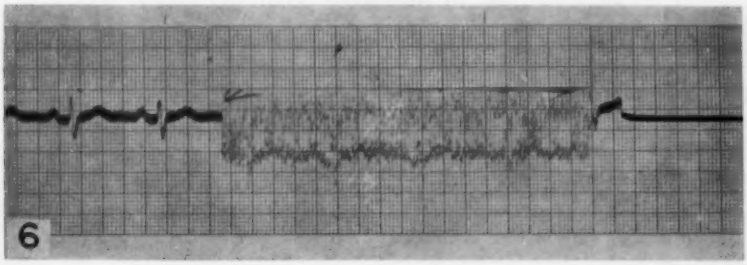


Fig. 5.—Comparison of effect of ocular stimulation versus tracheal stimulation (intubation).

the act of intubation of the trachea. This patient was a 31-year-old man undergoing surgery for correction of esotropia, with general anesthesia. Figure 5*A* and *B* show the effect of ocular stimulation in production of nodal rhythm and bradycardia, followed by disappearance of these effects with removal of the stimulus. Figure 5*C* represents the abolition of all effects by the retrobulbar injection, and, finally, Figure 5*D* demonstrates the lack of effect from tracheal intubation in the patient. It is indicated that ocular stimuli via the trigeminovagal reflex are more potent than are nonocular stimuli via the vagovagal reflex. This is of particular significance in view of the work of Reid and Brace,[19] who showed striking electrocardiographic changes upon irritation of the respiratory tract.

In an attempt to investigate the cardiac effects of surgical diathermy applied to the sclera, it was found that the diathermy machine set up such severe alternating-current interference that the electrocardiographic marker went out of control (Fig. 6). However, no clinical arrhythmia was observed (by palpation and auscultation) following scleral diathermy.

Fig. 6.—Effect of surgical diathermy applied to sclera.



Of greatest importance to us is the observation that, in the 12 cases in which a retrobulbar injection of 1% lidocaine (Xylocaine) or 2% procaine with epinephrine and hyaluronidase was performed following the demonstration of significant and reproducible electrocardiographic changes, complete abolition of the cardiac changes was accomplished in 100% of the cases. The effect usually occurred within one minute, but in every case it was observed within 90 seconds following the retrobulbar injection (Fig. 1). Finally, it is to be stated that in none of the cases studied was the retrobulbar injection found to produce any undesirable effect which made the operation more difficult to perform, or which might predispose to surgical errors in extraocular muscle surgery or retinal detachment operations.

Comment

Cardiac arrest has been of great and tragic interest since the first case of heart stoppage during anesthesia occurred, in 1848, in a 15-year-old girl anesthetized with chloroform for removal of an infected toenail. It has occurred in the young and the aged and with both local and general anesthesia. Its incidence in general surgery is stated to be about 1 in 1500 cases, while its incidence in ocular surgery has not, to our knowledge, been reported in the literature. We are, therefore, indebted to the surgeons-in-chief in eight of the larger ophthalmic institutions in the United States for supplying the information that leads to the conclusion that cardiac arrest occurs once in approximately 3500 eye operations.

It is thus seen that cardiac arrest is a real problem in ophthalmology, as will be sadly attested by any colleague who has experienced its devastation in his operating room. In consideration of its causes, fear is believed to be a significant predisposing factor, and known activating factors are simple reflex changes, hypoxia, hypercapnia, and sensitivity to or overdose of drugs (for reviews see Snyder, Snyder, and Chaffin [20] and Sealy, Young, and Harris [21]). It is surprising that with the vast knowledge concerning the oculocardiac reflex so little attention has been devoted to its study during actual ocular surgery.

The cases in the present series were operated upon under optimal conditions, with every precaution being taken to eliminate fear, hypoxia, hypercapnia, and drug toxicity. Nevertheless, 30% of the cases developed significant electrocardiographic changes, and two cases had transient cardiac arrest. This evidence, then, points strongly toward the paramount importance of the simple reflex changes in the production of the heart disturbances. Because of this evidence, our thinking early turned towards ways of blocking this reflex, and the action of the atropine-series drugs was first considered. In general surgery the parasympatholytic effect of atropine is well known, but this drug does not appear capable of complete abolition of the oculocardiac reflex, for Palm and Strömblad [17] found it ineffective in animals and Sorenson and Gilmore [18] observed that premedication with atropine in 7 of their 17 cases did not prevent clinical arrhythmia. Since the latter authors did find that intravenous atropine could block the reflex, and since they used no atropine by any route in 10 of their 17 cases, perhaps our consistent use of an atropine-series drug may explain, in part, our inability to confirm their production of clinical arrhythmia in every one of their 17 cases. An atropine-series drug was used in 48 of our 50 cases, the two exceptions being an 8-month-old child undergoing nasolacrimal duct probing, and in whom no oculocardiac reflex was elicitable, and the 48-year-old woman in whom (Fig. 4*B*) temporary cardiac arrest occurred. Despite our almost routine use of atropine, significant electrocardiographic changes occurred in 30% of our cases. It is thus apparent that an atropine-series drug may be helpful in decreasing the cardiac arrhythmias, but it cannot be relied upon for complete abolition of the danger.

If we pass, then, to the consideration of the retrobulbar injection as an oculocardiac reflex blocking agent, it appears that there have been only three cases reported, and these were employed to treat already-existent bradycardia resulting from postoperative orbital hematoma. Thus, Magitot and Bailliart,[22] 35 years ago, and later de Treigny,[11] used an orbital injection of procaine, and Michaud [16] used atropine by retrobulbar injection. Palm and Strömblad [17] reported the abolition of the oculocardiac reflex in experimental animals by a retrobulbar injection of lidocaine, although they did not investigate stimulation of the extraocular muscles. The evidence developed in the present communication, therefore, represents the first demonstration in man of complete and unfailing prevention, by the retrobulbar injection, of the electrocardiographic changes brought about by ocular stimulation of the trigeminovagal reflex. Because of this demonstration, it is recommended that a retrobulbar anesthetic injection be made a routine safeguarding procedure in every operation for strabismus, retinal detachment, or enucleation of the eyeball.

Summary

The cardiac effect of many ocular surgical maneuvers has been investigated in the operating room by means of serial electrocardiography.

In the 50 cases studied, significant electrocardiographic changes were induced by ocular stimulation in 15 cases, or 30%.

The significant electrocardiographic changes induced were the appearance of nodal rhythm, marked bradycardia, and, in two cases, temporary cardiac arrest.

The ocular stimuli found to produce these changes were digital pressure upon the globe, manipulation of the extraocular muscles, and direct pressure upon the tissue remaining in the orbital apex after enucleation.

These changes occurred under either local or general anesthesia.

Evidence has been adduced which indicates that ocular stimuli are more provocative of these electrocardiographic changes than are respiratory tract stimuli, i. e., the oculocardiac reflex is more sensitive than the pulmonocardiac reflex.

Complete abolition of these electrocardiographic changes induced by ocular surgery was accomplished in every instance by a retrobulbar anesthetic injection.

It is thus recommended that a retrobulbar anesthetic injection be made a routine safeguarding procedure in every operation for strabismus, retinal detachment, or enucleation of the eyeball.

The personnel of the operating room and the electrocardiographic laboratory of the Mount Sinai Hospital of Greater Miami cooperated in this study.

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Iridencleisis in Buphthalmia

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It is always salutary for himself, and possibly interesting to others, for a surgeon of some experience and assumed respectability to review the results he is getting from his treatment, surgical or otherwise, of any condition. This is particularly so if the condition is one of some etiological complexity, inevitably demanding surgical interference, and not hitherto susceptible of practically certain relief by one particular operation. Such a condition is buphthalmia. Recently one of my Registrars, noticing that I usually chose one particular operation for this condition, and presumably favorably impressed with the results, took the trouble to go through the notes of the cases of buphthalmia which had been in my care since the war, and to summarize the results. It is his interest and enthusiasm which has thus stimulated the production of this paper.

One must turn aside for a moment on the question of terminology. This disease was discussed at length in Chicago in 1954 under the title of congenital glaucoma, which was applied to "those cases of increased intraocular pressure in which the decreased aqueous outflow is due to defective embryological development of the eye." Those cases presenting within the first three years of life are called infantile glaucoma; those presenting later are called juvenile—all, however, presumably "congenital." There is probably some false grouping here, and certainly there is some pathological question-begging; a more fundamental objection to the term is that the condition is not always congenital. Kluyskens [10] gets over this objection by referring the adjective to the cause—a congenital malformation—and not to the disease; this makes the reason for the term clear but does not really dissipate its fundamental inaccuracy. My friend Robert Shaffer [22] says that when the disease manifests itself in very early life the resultant distended eyeball gave rise "in bygone years" to the "picturesque term buphthalmia." Well, I am not particularly "picturesque," but I am rapidly approaching the "bygone," and I think buphthalmia is a useful term which breaks no bones and implies no false pathology, and I have used it in my title. There are other examples of picturesque terms which similarly justify themselves. The familiar granuloma of the lid may be termed a Meibomian or tarsal cyst by some, who thereby unwittingly perpetuate a pathological falsity by implying that the lesion is primarily cystic, but those who apply the term chalazion use a term whose meaning is plain to all and carries no pathological solecism, for they are safe in assuming that no one will translate literally the Greek word which means, of course, a hailstone. I use the term buphthalmia to describe a condition of raised intraocular tension arising in early life, giving rise to expansion of the globe, and being almost certainly due to a congenital malformation impeding the drainage of aqueous humor.

There is incontrovertible evidence for the existence of this congenital malformation, and the etiological theory which derives from it is strengthened by the frequent coexistence of other skeletal malformations and maldevelopments and such conditions as congenital heart disease and mental defect. While the iris is commonly normal in itself, though frequently abnormal in its origin from the ciliary body, there is a fairly high proportion of cases in which it is absent altogether—a state of affairs which very considerably modifies the outlook for surgical relief. I have, on one occasion, on attempting to perform an iridencleisis for a

case of buphthalmia in which the cornea was opaque, found to my dismay that no iris was present at all and was thus forced to adopt an alternative surgical procedure.

From the variety of operations which used to be, and indeed still are, practiced for the relief of buphthalmia, it is evident that the condition presents a therapeutic problem and that no one operation is sufficiently often successful to be invariably adopted. It is further to be noted that here, as elsewhere, the operation that gives the best results in any surgeon's hands is the operation for that particular surgeon to do. The varieties of possible operations are endless; nowadays it is usually one of four that is carried out.

Trephining—still the operation of choice of many of us for chronic simple glaucoma—has its advocates. Mengel [14] speaks well of it, as does Pesme,[17] who emphasizes what I think is generally recognized—that operation must be undertaken in every case, preferably between the third and sixth month. Cyclodiathermy—surface or perforating—is very popular (Calmettes et al.,[6] Forbes,[8] Thomas et al.,[25] Algan,[1]), having the reputation of being less liable to cause immediate and remote complications and also being of value if surgery has failed or if the time for a fistulizing operation has passed. The only comment which at once comes to mind is that, in a condition indisputably due to inefficient drainage of aqueous, an attempt to improve this drainage would be a more physiologically appropriate endeavor than one aimed at reducing the production of aqueous. Algan [1] describes his treatment of 15 cases by perforating cyclodiathermy and claims 88% successes; his comparative figures are derived from other surgeons' results, and therefore the comparison is hardly a true one. An adverse comment upon the expression in percentages from so small a total as 15 would of course be met by the answer that we are, after all, dealing with a rare disease.

Goniotomy has become exceedingly popular of recent years, though it is of course no new thing. Tailor,[24] in 1891, and de Vincentiis,[7] in 1893, both described the operation of incision of the tissues of the angle of the anterior chamber for the condition of glaucoma in infancy. It obviously gives good results in some cases, in the hands of some surgeons. I would say in my characteristic conservative fashion that goniotomy is so far not acceptable without question just because its general adoption is so comparatively recent. I hold that it is too early to draw a firm conclusion as to its value. There are also good anatomical reasons for scepticism over the possible value of goniotomy in buphthalmia. All are agreed that the canal of Schlemm is often absent in buphthalmos (McArevey,[11] Young,[28] Wexler and Kornzweig,[27] Sugar,[23] François,[9] Reis,[18] Seefelder,[19] Kluyskens,[10] and many others), and I have on more than one occasion satisfied myself by the examination of serial sections, when the opportunity has arisen, that this channel is nonexistent. It is therefore difficult to see why an incision into the angle—malformed as that may be—could provide a permanent means of draining the chamber if the incision did not open the angle into some preformed and potentially adequate drainage channel. I have not adopted the operation myself. My reason for this at the outset was that I could not bring myself to scratch, blindly if hopefully, in a portion of the eye not within my view and that by the time the operating gonioscope arrived I was disappointed at the results I had seen. I have therefore continued to perform iridencleisis, and it is these circumstances which naturally induced me to review my results.

I am further very far from impressed when I am told—as I have often been—that if there is any doubt whether the condition of the angle will permit drainage after goniotomy, or if the canal is certainly absent, the incision may be carried through the limbal sclera into the subconjunctival region and drainage may occur subconjunctivally. This is, I submit, a misuse of the term

goniotomy as generally accepted; the operation is nothing more nor less than limbal puncture—a by no means useless maneuver which one's seniors were performing regularly at the time one was beginning to learn the subject and which is still extensively and successfully practiced.

It is indisputable that goniotomy has a high record of success in some hands; one has only to study the results of Barkan [3] to appreciate this. This procedure is enthusiastically advocated also by Scheie,[20,21] McKinney,[12] Bietti,[5] and many others, and it is certainly not my intention to question their statements or to cast any doubt upon the excellence of their results. One good reason for keeping an open mind is that the factors influencing operative success in buphthalmia are many and varied, and even one simple one such as the age of the patient may well be of immense importance in this connection. In the consideration of a subject such as this one must always bear in mind that there is a multitude of circumstances influencing any argument and that any conclusions must be drawn with due regard for variations—recognized or unsuspected—in such circumstances.

Iridencleisis is favored still by many (Bailliart,[2] Weekers [26]) and is said to carry less risk of damage than trephining. This is an important point, since it is my experience that the buphthalmic eye is less able to withstand interference than an eye not so affected—an opinion held by many observers. It is by no means rare to find that a fistulizing operation has been followed by subluxation of the lens, with or without consequent partial or total opacification; this has occurred in my experience after iridencleisis and has served to temper somewhat my enthusiasm for this procedure. The lack of likelihood of this complication is a strong argument in favor of goniotomy.

My actual technique for the operation is as simple as it is possible to make it. With use of a general anesthetic, and with the eye rotated downward by an assistant by means of Colley's forceps applied at the limbus at 6 o'clock, I turn down a conjunctival flap. An incision is made at the limbus some 4 mm. long by a keratome, and every endeavor is made to prevent sudden and exuberant loss of aqueous, with consequent danger of dislocation of the lens. The iris is grasped and pulled up into the wound, and a radial incision is made into it. One pillar is replaced, and any excess of the other is trimmed off after it is seen that it is firmly in the wound. A suture is not placed, and often a bandage is not applied; cardboard splints applied to the baby's arms are a safer protection to the eye than any cover and cause no distress. Atropine is used postoperatively.

The number of patients reviewed in this survey is 14, involving 20 eyes, of which 14 were submitted to iridencleisis. This is admittedly not a very large number, but the condition is rare, and in my country we cannot pretend to command such numbers as you do over here; further, I thought it advisable for many reasons to restrict my observations to my own personal cases. It is not my intention to go in any detail into other than the operative side of this series; the symptomatic, etiological, preoperative, and other factors of the disease have been ably and amply reviewed elsewhere on more than one occasion. In any such review, however, those aspects should be mentioned which may have a statistical value. For instance, there were eight males and six females, which accords with previous reports of proportion; less than half were bilateral (six against eight), whereas it is usually stated that some two-thirds of the cases are bilateral.

Some of the cases in this list need mention for special features, the presence of which in four cases prevents their inclusion in a review of the results of iridencleisis.

Case 2, in addition to the eye trouble, had nevus flammeus and club foot. It was possible to follow her for only two years after operation, as the family left London.

Case 5, though to be included in the review, might be discarded on the grounds that one was not given a fair chance with her. At the age of a week or two a goniotomy was performed on each eye by the surgeon then in charge; the parent was told there was nothing more to be done, that the child need not be seen again, and that her best action was to place the child at once in a home for the blind—a strange and cavalier attitude to a difficult but surely not inevitably hopeless problem. The baby was brought to me at 7 months; the vitreous of the right eye contained a whitish mass and there had obviously been low-grade infection. Iridencleisis was technically impossible at the first attempt, and so no more than limbal punctures were done; the right eye shrank, but subsequent iridencleisis in the left eye lowered the tension, and the child had obviously reasonable vision now at 6 years, though some lens opacity has developed.

Case 6 may not have been a true unilocular case, since the left eye had a large cornea though the tension was normal. The right tension was reduced to normal by iridencleisis and remained so eight years afterwards.

In the Case 7 anesthesia was followed by collapse and death—a reminder even from this short series that, as Barkan[3] emphasizes, these children need especial care in general examination before surgery is attempted.

Case 8 had had two operations on each eye before the age of 8 days—surely rather an overenthusiastic approach; further, the operations were wide iridectomies. The child was sent to me at the age of 2 years for a second opinion, and I advised further operation, but nothing was done. The child was brought to me again four months later, being then on the blind register. Iridencleisis reduced the tension, though I fear that it was then too late.

It proved impossible to perform iridencleisis on the left eye of Case 10; Herbert's sclerotomy was done and proved successful.

Fig. 1.—Age at operation.

Case 11 was a mental defective with aniridia. Many punctures and Herbert sclerotomies were done, but the child was sent to a Sunshine home with the tension still full at the age of 10.

Case 12 had had cyclodialysis done on his one affected eye before he was brought to me at the age of 6. I did a cautery puncture and followed this two months later by cyclodialysis. At the age of 23 vision in this eye was 6/18 and tension was normal.

Cases 7, 8, 11, and 12 will all be omitted for the reasons given.

Iridencleisis on both eyes in Case 13 left the left eye still hard; iridencleisis six months later reduced the tension, which was still normal five years afterward. The left lens was observed to be subluxated, with commencing opacification, which may explain the necessity for the second operation.

We are left, then, with 10 cases yielding 14 eyes operated upon by iridencleisis. It is important to record the age at operation, and this is shown graphically in Figure 1, which of course omits the two cases in which iridencleisis was not performed (Cases 11 and 12) but includes two omitted from the final survey because no follow-up was possible. The length of follow-up in the 10 cases is shown graphically in Figure 2.

In 1 of these 10 cases, as I have just mentioned, the operation had to be repeated, and the tension was then successfully

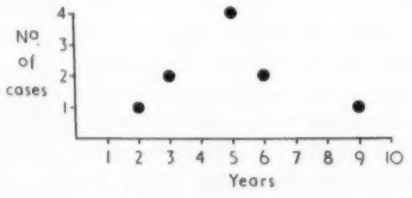

Fig. 2.—Follow-up.

brought to normal. All the others were reduced by the one operation, and all have remained normal up to the last time the patients were seen. It is not justifiable with such small numbers to indulge in percentages, but if one does the results may be summarized as a 100% success for iridencleisis and 91% for a single iridencleisis operation.

A word should be added about criterion of success. Visual acuity is usually quite impossible for reasons of age; further, irreversible corneal changes may have occurred before the patient is seen, which make this criterion inapplicable and unfair. Tonometry would appear to offer a good criterion, but I submit that it does not. Digital examination is the only reliable method we have of estimating tension in this disease, and in experienced hands it is to be trusted. A more chancy instrument than many realize at all times, I am convinced that in buphthalmia the tonometer is completely unreliable, probably because of altered corneal curvature and resilience. I would not trust even the same instrument on the same eye in the same hands at different times, for I, with others, have had experience of variable results on the pathological infantile eye. Incidentally, I take this opportunity of expressing the view that too much emphasis can easily be laid upon tonometry, even in adult glaucoma; it is an obscure symptom complex of which ocular hypertension is but one feature, and tonometry is but one way, and a doubtfully accurate way, of estimating that. The idea of distributing tonometers in all medical directions for the indiscriminate use of the inexperienced is one needing careful thought and planning before execution, lest it do more harm than good. Juvenal has expressed my feelings for me: *Quis custodiet ipsos custodes?*—"Standardize tonometers as you may, who is going to standardize the tonometrists?"

It will be recalled that the final conclusion of the Chicago symposium on congenital glaucoma was that goniotomy was the operation of choice. With this I have no quarrel, but I have been bold enough to point out some reasons why I would not always expect good results from goniotomy. I hasten to add that were I in the privileged position of operating, as Otto Barkan does, on 212 eyes, I would not expect my figures to be as favorable as they are in my small series; I feel confident, however, that they would equal, or possibly surpass, the 77% success of goniotomy, which, incidentally, had to be repeated once in half the cases and three, four, and even five times in some.

Iridencleisis is a valuable and successful method of treatment of a rare, serious, and difficult infantile condition, the relief of which necessitates surgery, which surgery demands care in choice and extreme delicacy in execution.

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Use of a Thiophosphinyl Quaternary Compound (217-MI) in Treatment of Glaucoma

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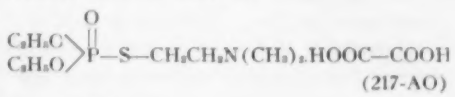
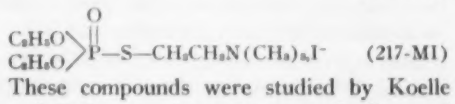
Anticholinesterase agents have been used for the control of intraocular pressure for many years. The first was physostigmine (eserine), an alkaloid obtained from the Calabar bean. In 1863 Fraser noted that physostigmine caused pupillary constriction, and Laqueur in 1875 employed it for the treatment of glaucoma.[8] At that time it was noted that undesirable side-effects occurred with the drug. Ever since, an intermittent search for a more potent, less irritating anticholinesterase agent has been under way. A variety of cholinesterase agents have been tried. These include those which form a reversible union with cholinesterase as does physostigmine, namely, neostigmine and *o*-neostigmine and *p*-neostigmine. Other anticholinesterase agents inhibit cholinesterase irreversibly and include tetraethylpyrophosphate (TEPP), tetraisopropylpyrophosphate (TIPP) hexaethylpyrophosphate (HEPP), *p*-nitrophenyldiethylphosphate, (Mintacol), parathion (*o,o*-diethyl-*o*-*p*-nitrophenyl thiophosphate), isoflurophate (DFP, Floropryl), and tabun (dimethylamidoethoxy-phosphoryl cyanide).[1] Of these, physostigmine, neostigmine (Prostigmin), and isoflurophate have stood the test of time as useful agents in the therapy of glaucoma. Isoflurophate is considered to be the most potent of these.[3,5-7]

Recently there has become available a tertiary and a quaternary form of an extremely potent thiophosphate anticholinesterase agent. The chemical structure of the tertiary form, 2-diethoxyphosphinylthioethyldimethylamine acid oxalate (217-AO; 217-acid oxalate), and that of the quaternary form, 2-diethoxyphosphinylthioethyltrimethylammonium iodide (217-MI; 217-methyl iodide), are as follows:



(217-AO)



(217-MI)

These compounds were studied by Koelle and Steiner to determine central effects of these drugs after peripheral administration.

Following the intravenous administration of both forms to rabbits, the tertiary form produced 90% inactivation of the total brain acetylcholinesterase, while the quaternary derivative caused no measurable inactivation of the brain acetylcholinesterase. There was noted, however, a marked apparent inactivation of the acetylcholinesterase of the entire brain following the injection of 1/100 of the previous dose of 217-MI into the lateral ventricle. With this evidence that the quaternary agent is a potent anticholinesterase agent that does not permeate the blood-brain barrier and therefore has no central effects, the investigation of the miotic and antiglaucomatous effects of this drug in the human eye was begun.

The quaternary form of the thiophosphate termed 217-MI is a white crystalline water-soluble compound.

The Effect of 217-MI on the Normal Eye

The compound 217-MI has a marked and prolonged miotic effect in man. In a series of 27 normal humans, 1 drop (about 0.06 cc.) of 0.5% 217-MI was instilled in the right cul-de-sac. Miosis began within 10 to 45 minutes and lasted 7 to 27 days. The

Wills Eye Hospital.

instillation of 217-MI in the normal eye was accompanied by symptoms of ciliary spasm, namely, blurring of vision and brow ache. In addition, slight circumcorneal injection was noted. The intraocular pressure rarely fell more than 2 to 3 mm. Hg (Schiøtz). No precipitous rises of ocular tension were encountered in these normal human eyes.

Stability

The compound 217-MI is stable in isotonic saline solution. When it is kept at 5 C the problem of deterioration can be totally disregarded. After 5 weeks at room temperature the solution retained 75% anticholinesterase activity; after 10 weeks, 62% activity; after 30 weeks, 55% activity remained. The storage stability of 217-MI in isotonic saline solution without preservative at room temperature is shown in Table 1.

TABLE 1.—*Storage Stability of 217-MI*

Storage Time, Wk.	Remaining Activity * in Sterile Solution Without Preservation at Room Temperature, %
0	100
10	62
20	56
30	55

*Ability to inhibit cholinesterase.

Effect of 217-MI on Homatropinized and Atropinized Eyes

The effects of 217-MI on eyes homatropinized with 4% solution four times in 2 hours and atropinized with 1% solution three times in 36 hours were studied in various age groups. Two and sometimes three instillations of 217-MI at 10-minute intervals produced partial miosis in the eyes treated. The accommodation was likewise partially augmented. These effects were greater in homatropinized than in atropinized eyes.

Use of 217-MI in Treatment of Glaucoma

Fifty-eight patients, with 101 glaucomatous eyes, were treated during a nine-month period. Every patient had been treated previously with other miotic agents, with or without supplementary acetazolamide (Diamox) therapy, before 217-MI was initiated. The patients treated were chosen at random, regardless of the type of glaucoma, from the clinics of Wills Eye Hospital. The 101 glaucomatous eyes treated are summarized in Table 2.

TABLE 2.—*Types of Glaucoma Treated*

	Eyes, No.
Wide angle	54
Narrow angle	31
Aphakic	12
Hemorrhagic secondary to central vein thrombosis	2
Secondary to dislocated lens	1
Secondary to corneal opacity	1
	101

Previous Therapy.—Previous therapy had consisted of a variety of miotic agents either singly or in combination with other miotics, as outlined in Table 3. Some patients had been treated with a single miotic three to six times daily, while others had received two and three miotics simultaneously four to six times daily. Thirty-three of the fifty-five patients received acetazolamide orally in dosages from 125 mg. twice a day to 250 mg. four times a day in addition to the miotic therapy outlined in Table 3.

Dosage.—The dosage of 217-MI varied in each individual case. Three concentrations were utilized, 0.5%, 0.25%, and 0.1%. In each individual case an attempt was made to use a minimum concentration with instillations as infrequently as possible and

TABLE 3.—*Previous Therapy*

Miotics Used			Patients, No.
1. Pilocarpine	1%-4%	3 times daily to 6 times daily	26
2. Pilocarpine + Physostigmine	1%-4% + 0.25%	4 times daily to 6 times daily	14
3. Carbachol	1.5%	4 times daily to 5 times daily	3
4. Isoflurophate	0.05% to 0.1%	once daily to 2 times daily	7
5. Pilocarpine + Physostigmine	4% + 0.25%	4 times daily to 6 times daily	8
Physostigmine oint.	0.25%	at bedtime	
			58

Acetazolamide: 33 patients (61 eyes) of the 58 patients received acetazolamide in dosage from 125 mg. b. i. d. to 250 mg. q. i. d. in addition to miotic therapy

TABLE 4.—*Summary of Results*

Type of Glaucoma Treated	Controlled on Previous Medication, No. of Eyes	Uncontrolled on Previous Medication, No. of Eyes	Controlled by 217-MI, No. of Eyes	Uncontrolled by 217-MI (Unsuccessful), No. of Eyes
Wide angle	28	29	45	12
Narrow angle	19	9	17	11
Aphakic	5	7	10	2
Secondary to central vein thrombosis		2	1	1
Secondary to dislocated lens		1		1
Secondary to corneal opacity		1		1
Total	52	49	73	28

	Eyes, No.
Controlled on previous therapy and on 217-MI therapy	31
Uncontrolled on previous therapy and controlled on 217-MI therapy	42
Controlled on previous therapy and uncontrolled on 217-MI therapy	20
Uncontrolled on either previous therapy or 217-MI therapy	8

yet to control the intraocular tension. Instillations varied from twice weekly to twice daily.

Criteria of Control.—Glaucoma was considered controlled if the ocular tension was below 30 mm. Hg (Schiøtz) and if there was no further loss of visual field, no consistent decrease in coefficient of aqueous outflow, and no visible evidence of progression of disease by gonioscopy, biomicroscopy, or ophthalmoscopy.

Results.—The results are summarized in Table 4. One hundred and one eyes were treated with 217-MI therapy. In 73 eyes glaucoma was successfully controlled with 217-MI therapy. Twenty-eight eyes were recorded as failures; in twelve of the failures the intraocular pressure was well controlled with 217-MI; however, the drug was discontinued because of untoward side-effects. In 16 eyes 217-MI was discontinued because of its failure to control ocular tension. In 2 of these 16 eyes a paradoxical rise in ocular tension was noted; one was a case of glaucoma secondary to central vein thrombosis; the second was a case of chronic narrow-angle glaucoma in which an acute congestive phase developed, and a basal iridectomy was performed. The results of the treatment with 217-MI compared with previous therapy are outlined in Table 4.

In 31 eyes glaucoma was controlled both by previous therapy and by 217-MI; glaucoma in 42 eyes which was uncontrolled by previous therapy was controlled by 217-MI; in 20 eyes glaucoma was controlled by previous therapy and treatment with 217-MI was considered a failure. Glaucoma in eight eyes was uncontrolled with either previous therapy or 217-MI.

The percentage of successful cases treated with 217-MI was much higher in wide-angle glaucoma and aphakic glaucoma than in narrow-angle glaucoma.

Diurnal fluctuations in intraocular pressure were notably less in the patients whose glaucoma was controlled with 217-MI than with previously employed miotics.

Effective Concentration.—The most effective concentrations of 217-MI in the treatment of glaucoma were 0.25% and 0.1%. These concentrations gave optimal therapeutic effects of the drug with minimal side-effects. Higher concentrations of the drug produced no greater ocular tension effect, only more profound side-effects.

The Untoward Effects Noted.—The untoward effects noted, in order of their frequency, were (1) brow ache and (2) dimness of vision (14 eyes); (3) paradoxical rise in pressure (2 eyes), and (4) nausea (complained of by one patient).

To the present date there have been (1) no detachments noted; (2) no local sensitivity; (3) no follicular conjunctivitis, as reported with physostigmine therapy; (4) no pseudomembranes; (5) no tear duct obstructions.

The symptoms varied from patient to patient; they were less frequent and less severe with the lower concentrations. Ciliary injection, which was very prevalent when 217-MI was used on normal eyes, was much less prevalent when it was used on glauco-

matous eyes. Aphakic eyes were less troubled by side-effects than phakic eyes. Fourteen eyes of treated patients had the above symptoms to a severe degree, and in twelve of these the drug had to be discontinued.

Tonography.—Studies with tonography suggested decrease in resistance to outflow of aqueous humor during therapy in the majority of eyes treated with 217-MI, as compared with former therapy.

Comment

The compound 217-MI is a powerful anticholinesterase agent which does not permeate the blood-brain barrier. It is water-soluble and stable, which adds to its applicability. Isofluorophate, a comparably potent anticholinesterase, is stable only in anhydrous vehicles, such as peanut oil. Although 217-MI is not the ideal miotic for all antiglaucomatous therapy, it has definite attributes which entitle it to a significant trial in our antiglaucomatous armamentarium, especially in the treatment of wide-angle and aphakic glaucoma. It will lower intraocular pressure in some eyes in which pressure has not been previously controlled by combinations of local miotics and systemically administered acetazolamide. The duration of action favorably influences diurnal fluctuations and reduces the necessity of frequent instillations. As with other strong miotics, its use is accompanied by undesirable side-effects.

Summary

The compound 2-diethoxyphosphinylthioethyltrimethylammonium iodide (217-MI) controlled intraocular pressure previously uncontrolled by the combined therapy of other miotics and acetazolamide. It controlled intraocular pressure previously controlled by other miotics, but the dosage and frequency of administration were greatly reduced.

The duration of action of 217-MI is longer than that of any commonly used miotic and therefore may be useful in dampening the diurnal fluctuation.

The 0.25% and 0.1% concentrations of 217-MI solution in isotonic saline are stable indefinitely at 5 C and show slow diminution in activity at room temperature.

No instances of dermatitis and conjunctivitis due to repeated use of 217-MI were encountered.

The compound 217-MI has some untoward ocular side-effects in common with other strong miotics, namely, blurring of vision, brow ache, slight pain in the eye after initial administration, and paradoxical precipitous rise in intraocular pressure.

There were no obvious systemic effects following ocular administration of 217-MI.

The 217-MI used in these studies was provided by Campbell Pharmaceutical Company, 254 W. 31st St., New York 1.

Wills Eye Hospital, 1601 Spring Garden St. (30).

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A Surgical Approach to Total Xerophthalmia

Transplantation of the Parotid Duct to the Inferior Cul-de-Sac

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Introduction

The treatment of total xerophthalmia has always been a very difficult problem. Total xerophthalmia frequently occurs following exfoliative dermatitis, ocular pemphigus, and trachoma, which are associated with complete loss of function of the lacrimal gland and all accessory lacrimal tissue. Conservative treatment, such as topical instillation of artificial tears, blocking of the puncta, tarsorraphy, transplantation of buccal mucosa, and vitamin A therapy is usually unsatisfactory and generally gives only temporary improvement. The usual course of total xerophthalmia is corneal ulceration, epidermidalization, and keratinization of corneal epithelium; symblepharon, and corneal vascularization with progressive visual loss. Transplantation of the parotid duct into the lower conjunctival cul-de-sac offers a new approach towards the solution of this problem.

Comment

In 1950, Filatov [1] and his associates first reported the successful transplantation of the parotid duct into the conjunctival sac for the treatment of total xerophthalmia, and subsequently they have reported 80 cases. Additional cases of transplantation of the parotid duct have been reported by Katznelson,[2,3] Ao,[4] Yen and Lee,[5] Hsia,[6] Bulak,[7] Lao,[8] and others. No statistical evaluation of the results in these cases was found in the Russian literature; however, it was stated that there were no complications and that the results were good. Lao [8] in his paper reviewed the cases reported in the Chinese literature. There were 12 cases in his summary. In every instance there was an improvement in visual acuity following the parotid duct transplantation. In three instances he reported an excessive lacrimation following the transplant, but in one instance this corrected itself after a few months. In one case he reported a wound infection as a complication, and he stated that in two instances the duct was too short to reach the cul-de-sac and that deepening of the fornix was necessary in order to complete the transplantation successfully. There were two cases of entropion, graded as slight, which he attributed to traction of the duct on the cul-de-sac. No mention was made of facial paralysis or other serious complication, and in every instance a functioning result was achieved.

The American literature has been reviewed. No report of transplantation of the parotid duct into the conjunctival sac was found. In view of the successful results reported by the Russian and Chinese workers and the failure of conventional treatment in this disease, it was felt that the feasibility of the procedure should be investigated. Transplantation of the parotid duct was done successfully on four dogs, and a surgical approach was devised on the human cadaver.

From the Section of Ophthalmology, Department of Surgery, Crile Veterans' Administration Hospital.

The personnel in the animal house and in the medical illustration department of Crile Veterans' Administration Hospital cooperated in making this study possible. Miroslaw Komarjanski, M.D., U. S. Veterans' Administration Hospital, Brecksville, Ohio, translated the Russian articles, and Alice Lee, M.D., Mt. Sinai Hospital, Chicago, translated the Chinese articles.

Experimental Work

A. Surgical Procedure on the Dog.

After surgical preparation and with use of intravenous pentobarbital (Nembutal) anesthesia, a fine probe was inserted into the parotid duct to aid in the identification of the duct during its surgical isolation. An area of buccal mucosa 7 mm. in diameter surrounding and attached to the os of the parotid duct was incised and dissected free. The wide excision of buccal mucosa was made so that it could act as a funnel and form an extension of the parotid duct should it prove too short to reach the inferior cul-de-sac. Two 000 plain absorbable surgical (gut) sutures were then placed in diametrically opposed edges of the buccal mucosal flap.

An incision 2.5 cm. long was made through the skin of the cheek over the duct. The parotid duct was dissected free for approximately 3 cm. from its entrance into the mouth, back towards the partotid gland. The fine probe previously inserted into the parotid duct served as an aid in the identification of the duct during this dissection. A sharp-pointed scissors was then inserted through the base of the outer third of the inferior cul-de-sac, and the tissue was undermined superficially, the opening emerging in the incision on the cheek, thus creating a subcutaneous tunnel. A hemostat was then substituted for the scissors, and the sutures in the buccal funnel were grasped and drawn through the subcutaneous tunnel into the inferior cul-de-sac. The probe was removed from the parotid duct, and the sutures were drawn taut, raising the buccal funnel into the base of the lower conjunctival cul-de-sac, where it was sutured in place. The skin incision in the cheek and the buccal mucosal defect in the mouth were each closed with interrupted gut sutures. Postoperatively the animal was given 1,000,000 units of penicillin intramuscularly. No dressing was applied. The reaction to surgery was minimal, and the postoperative course, uneventful.

Fig. 1.—Parotid secretion dripping from dog's eye. This dog has had a transplantation of the right parotid duct into the inferior cul-de-sac. Following intramuscular injection of 5 mg. of pilocarpine per 10 lb. of body weight, there was a copious flow of parotid secretions through the transplanted duct.

B. Results.—Transplantation of the parotid duct into the inferior conjunctival cul-de-sac in the dog has proved to be a relatively easy and successful procedure. The function of the transplants has been verified by subcutaneous injection of pilocarpine, which is a very effective sialogogue, with little or no effect on lacrimal secretions. Following transplantation of the parotid duct to the inferior cul-de-sac, the subcutaneous injection of pilocarpine, 5 mg. per 10 lb. of body weight of the dog, produced a copious flow of parotid secretion into the inferior cul-de-sac (Fig. 1).

The parotid secretions were well tolerated, the cornea and conjunctiva appearing normal and healthy. Lacrimal drainage was adequate, inasmuch as only slight epiphora occurred. The first of these operated-on dogs has been observed over a period of three months, with satisfactory results.

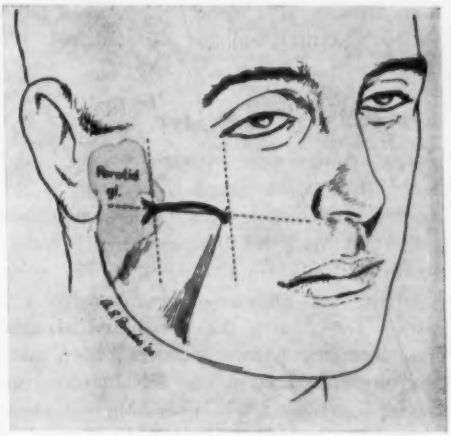

Fig. 2.—Surgical anatomy. A diagrammatic illustration of the surgical landmarks for localizing the portion of the parotid duct to be mobilized.

Fig. 3.—Surgical approach to parotid duct. A diagrammatic illustrations of the exposure of the parotid duct, utilizing the landmarks of Figure 2.

C. Cadaver Work.—To evaluate further the feasibility of the transplantation of the parotid duct for xerophthalmia, dissection was carried out on a human cadaver. The general anatomical details of the area can readily be found in any anatomy text and will not be discussed here. Pertinent surgical anatomy and landmarks are pointed out.

The parotid duct in man is found to lie along a line drawn from the base of the ear to the lower margin of the alae of the nose (Fig. 2). The parotid duct is approximately 7 cm. in length and arises as numerous branches within the substance of the parotid gland. At the anterior border of the gland or slightly in front of the gland the main duct receives a branch from the accessory portion of the parotid gland. In transplantation of the duct mobilization of the duct is not necessary beyond this point.

The entrance of the parotid duct into the mouth lies at about the point of intersection of a vertical line from just inside the lateral canthus of the eye with the horizontal line from the base of the ear to the lower margin of the alae of the nose (Fig. 2). The junction of the accessory duct with the main duct lies on this horizontal line approximately half way between the base of the ear and the entrance of the duct into the mouth.

The portion of the parotid duct to be utilized for transplantation, i. e., from the

Fig. 4.—Transplanted duct. A diagrammatic illustration of the transplanted parotid duct in the cadaver, showing the utilization of the buccal mucosal funnel.

junction of the main duct and the accessory duct to the entrance of the duct into the mouth, may be easily and accurately localized by use of the above-mentioned landmarks. An incision 2.5 cm. in length over this portion of the duct gives adequate exposure (Fig. 3).

The duct is easily recognized and isolated just beneath the buccal fat pad as it turns deep over the anterior edge of the masseter muscle. The only other major structures in this area are branches of the facial nerve and the transverse facial artery. The transverse facial artery gives no problem in this approach, since the incision is well below the artery. The incision roughly parallels the course of the branches of the facial nerve, and there would appear to be minimal damage to them. The parotid duct is of adequate length to reach the cul-de-sac, but utilization of a buccal mucosal funnel as described in the surgery of the dog is advisable in order to avoid tension on the transplanted duct (Fig. 4).

D. Production of Xerophthalmia.—In the interest of more complete evaluation of this problem an attempt to produce total xerophthalmia in dogs was done by excision of the lacrimal gland and superficial x-ray irradiation of the conjunctiva, with the globe shielded. To date results have been unsatisfactory, and further work along this line has been planned.

Comment

In the further consideration of the feasibility of doing this procedure on the human being it is well to consider the similarities of the parotid and lacrimal glands. The histological structures of the two glands are similar (Table). The secretions are continuously secreted. Saliva from the submaxillary glands is sufficient for mastication.

Comparison of Lacrimal and Parotid Secretions

	In Tears[9]	In Parotid Secretion[10,11]
pH	8.2	5.3–7.8
Total nitrogen, %	0.158	0.045
Ammonia nitrogen, %	0.003	0.0076
Chlorides, %	0.394	0.040
Total solids, %	1.8	1.59
Ash, %	1.05	0.81
Total protein, %	0.669	0.2–0.4

Summary

The conservative treatment for total xerophthalmia has been discouraging, but Russian and Chinese workers have suggested a new approach to this problem by transplanting the parotid duct into the conjunctival sac. They report many successful cases. To confirm the feasibility of this procedure, the parotid duct has been successfully transplanted into the conjunctival sac in four dogs. The operation has been worked out on the human cadaver, and a surgical plan has been devised.

Conclusion

In view of the original work done by Russian and Chinese workers on the transplantation of the parotid duct into the conjunctival sac, for treatment of xerophthalmia in man, and our success in confirming this procedure in dogs, it is believed that this operation may provide a solution to the problem of total xerophthalmia in man.

Addendum

On Nov. 29, 1956, transplantation of the parotid duct to the inferior conjunctival cul-de-sac was performed successfully on a patient with total xerophthalmia at Crile Veterans' Administration Hospital. The transplanted duct has been functional, and postoperatively there has been both subjective and objective improvement. An evaluation of this case will be reported in a subsequent paper.

5500 Ridge Rd. (29).

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Surgical Treatment of Total Xerophthalmia

Transplantation of the Parotid Duct to the Inferior Cul-de-Sac with Report of a Case

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Introduction

The use of parotid secretions as a continuous and satisfactory substitute for lacrimal secretions in total xerophthalmia was first advocated by Russian [1-3] and Chinese workers.[4-7] They have reported several hundred cases in which parotid duct transplantation to the inferior conjunctival cul-de-sac was performed successfully in total xerophthalmia. However, the literature contains little more than the fact that the transplantations were successful and beneficial, with no evaluation of the results.

We [8] have investigated the substitution of parotid secretions for lacrimal secretions by transplanting parotid ducts in dogs and observing these dogs over periods up to eight months. The parotid secretions were well tolerated by the eye and produced no untoward effects. The surgical approach was worked out on the human cadaver, and a surgical technique was devised.

Report of Case

A 24-year-old male Negro was admitted to the neurology service, Crile Veterans Administration Hospital, Cleveland, on Aug. 23, 1955, for treatment of post-traumatic epileptiform seizures resulting from a skull fracture in 1950. Ten days prior to his admission to Crile Veterans' Administration Hospital, the patient had been started on methoin (Mesantoin) therapy. At the time of admission the patient was febrile and had a morbilliform rash on the chest. He subsequently developed a very severe exfoliative dermatitis, and, after a stormy course, his condition finally improved on cortisone therapy. Following the exfoliative dermatitis the patient developed bilateral xerophthalmia. The Schirmer test revealed no wetting of the filter paper. There was a vascular injection of the conjunctiva and recurrent corneal ulcerations, and the patient complained of severe ocular pain. Ankyloblepharon had occurred in the area of the lacrimal puncta, completely occluding them. The patient was given local antibiotic ointments, with patching of the eyes, until the corneal ulcerations healed, and therapy was then continued with various combinations of methylcellulose, antibiotic drops, and bland ointments. In spite of continuous therapy, recurrent corneal ulcerations occurred; the eyes were constantly injected and painful, and the patient required almost constant hospital care. The visual acuity gradually decreased to 20/200 in the right eye and 12/200 in the left eye. Corneal scarring occurred, and a vascularized pannus developed in the left eye.

Treatment.—In view of the progressive visual loss which was occurring in spite of conservative treatment, it was decided to attempt transplantation of the parotid duct to the inferior conjunctival cul-de-sac of the left eye, which was the more seriously involved and the least satisfactorily controlled. On Nov. 29, 1956, the left parotid duct was transplanted into the inferior conjunctival sac.

Surgical Technique.—With use of general anesthesia, a 2.5 cm. skin incision was made. This incision began directly below the lateral canthus on a line extending from

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the ala of the nose to the base of the ear and extended posteriorly along this line approximately one-half the distance to the base of the ear (see Fig. 2 of preceding paper in this issue [8]). The parotid duct was identified and isolated as it passed over the masseter muscle. A rubber dam was passed beneath the parotid duct to facilitate handling it. The mucous membrane in the mouth was then incised with a Bard Parker knife, leaving about 1 cm. of mucous membrane around the os of the duct. The mucous membrane around the os of the duct was used to lengthen the duct when the transplant was made. The mucous membrane incision was then carried through the buccinator muscle, freeing the duct from the mouth and allowing it to be pulled out through the skin incision. The duct was then freed posteriorly by means of sharp dissection to the junction of the main and accessory ducts, thus mobilizing the duct as much as possible for the transplantation. Plain 000 absorbable surgical (gut) sutures were passed through the edges of the mucous membrane around the os of the duct, to be used to pull the duct through the subcutaneous tunnel and to suture the mucous membrane to the conjunctiva. There is a muscular thickening of the buccinator around the os of the duct, and this excess tissue was trimmed prior to transplantation.

With scissors an opening was made through the conjunctiva in the lateral one-third of the inferior cul-de-sac and extended subcutaneously to exit in the posterior portion of the skin incision. The conjunctival incision in the cul-de-sac was made well back in the cul-de-sac, to allow mobilization of the conjunctiva in making the anastomosis with the transplanted duct without placing traction on the lower lid. It was necessary to utilize the conjunctiva in making the anastomosis, because the transplanted duct was several centimeters too short to reach the cul-de-sac. A hemostat was then passed from the conjunctival incision in the cul-de-sac down through the subcutaneous tunnel into the skin opening,

Transplanted parotid duct and lacrimal sac. The transplanted parotid duct was too short to reach the inferior conjunctival cul-de-sac primarily. Therefore, it was necessary to utilize conjunctiva and oral mucous membrane to lengthen the duct, as illustrated. Conjunctiva was also utilized to lengthen the transplanted nasolacrimal sac. Slight conjunctival traction is of value here to produce a funnel effect to aid gravity in removing the excessive secretions.

and the gut sutures previously placed in the mucous membrane surrounding the os of the duct were grasped and drawn through the tunnel. The parotid duct was then drawn through the subcutaneous tunnel, with care to make sure that its course was smooth and that no twists were present. A lateral canthotomy and freeing of the lower lid was necessary, because the anastomosis was made beneath the skin approximately 0.5 cm. below the cul-de-sac, as is illustrated in the Figure. The mucous membrane around the os of the duct was then sutured to the mobilized conjunctiva of the cul-de-sac. The canthotomy, the oral mucous membrane defect, and the skin incision were repaired with interrupted black nonabsorbable surgical (silk) sutures. A light dressing was applied.

Postoperative Course.—The postoperative course was uneventful; the skin incision healed with only a small linear scar. There was a mild lower facial weakness for ap-

proximately two weeks postoperatively, but this cleared completely. The patient had almost immediate relief of all pain and discomfort in the eye, and the vascular injection of the conjunctiva decreased. The visual acuity improved rapidly and in two weeks changed from 12/200 to 20/60. The flow of parotid secretions into the eye was excessive, particularly during eating, which produced marked epiphora. The patient felt this to be a relatively minor inconvenience. It must again be pointed out here that this patient had no lacrimal drainage system, owing to ankyloblepharon which occluded the lacrimal puncta. In a patient with a normal lacrimal drainage system, the excess parotid secretion might not be so great a problem.

After a month the patient developed a traumatic keratitis from continually wiping the eye to remove the excessive secretions. An attempt was made to reduce the flow with tincture of belladonna in increasing doses, but this was only partially successful. It became apparent that some form of lacrimal drainage would be necessary. Therefore, on March 8, 1957, the patient was taken to the operating room and, through a skin incision over the side of the nose, the lacrimal sac was isolated and completely freed. An opening was made in the cupola of the sac, and the nasolacrimal duct was found to be patent. The anterior lacrimal crest was then removed to facilitate working. The mobilized lacrimal sac was then transplanted laterally and the cupola of the sac was sutured to the conjunctiva of the inferior cul-de-sac, as illustrated in the Figure. A polyethylene splint was put into place. This created an epithelial-lined tube from the inferior cul-de-sac to the nose through the nasolacrimal duct. The skin incision was closed with interrupted black silk sutures. This drainage has worked well postoperatively, and the patient now has minimal epiphora. The keratitis has cleared. The eye continues to be comfortable, and the visual acuity has remained at 20/60.

Summary

The successful treatment of a case of total xerophthalmia by means of transplantation of the parotid duct is reported. The surgical technique is described. The postoperative result and complications are discussed. A surgical treatment of epiphora is presented, which utilizes the lacrimal sac.

Conclusion

Surgical treatment of total xerophthalmia by transplantation of the parotid duct has been demonstrated to be a feasible procedure.

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New Aspects of Ophthalmoneurologic Diagnosis

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Introduction

During the past several years sufficient experience has been gained in the technique of electromyography of the human extraocular muscles to fairly appraise its usefulness.[1-10] As a research tool it is of inestimable value in the analysis of the innervational basis of normal and disordered motility. Fundamental insights are being gained in the mechanisms maintaining normal extraocular muscle balance, so essential to binocular single vision. The sources of tonic innervation of eye muscle, although not well understood, now have become considerably clearer and may eventually be fully revealed in the course of analytic studies. The complex interrelationship of versions and vergences is being clarified, permitting a new understanding of the manner in which central adjustment of opposing sources of innervation is brought about. Through the study of varied types of pathologic involvement of the extraocular muscles it has been possible to make certain preliminary judgments concerning the nature of the lesion and the level at which different forms of innervation are developed. These may be confirmed or disproved by animal experiments currently in progress.

In the field of neurogenic disorders, electromyography has proven itself a new and important diagnostic tool, whose potentialities may not be fully realized for years. The practical value of electromyography at the present time lies largely within this group of conditions and is, therefore, equally of interest and importance to neurology and to ophthalmology. In the field of comitant disorders of childhood, the technique, as yet, has not achieved comparable utility, largely owing to the impracticability of inserting electrodes in young children. This is not an absolute drawback, since we have been able, in selected cases, to accomplish this without any difficulty. Our youngest patient was 1½ years of age. A phlegmatic child or one moderately sedated may afford little difficulty in this regard. Hyperkinetic children are not suitable subjects for electromyography. It is highly significant that our findings in young children do not differ in any major respect from those in adults, and it is quite reasonable to apply the findings in adult patients to comparable conditions of childhood.

Electromyography is not a tool for office use, nor is it suitable to any and all institutions. It should be conducted in centers where adequate technical aids and personnel are available—electronic consultants, electromyographers, and/or neurophysiologists. Such a center can afford invaluable diagnostic help to practitioners in both ophthalmology and neurology. It is not a field for the dilettante. There are many pitfalls in interpretation and technique which only experience and guidance can help one to avoid. Simply because a tracing appears in black and white does not mean that the truth is also a simple matter of black and white. Artefacts of needle position, of electronic instrumentation, of patient movement, etc., may obscure the true state of affairs. Despite these difficulties, in experienced hands the technique affords many clear-cut objective evidences of the innervational situation

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in eye muscle. It should be emphasized that similar cautions are currently urged by professional electromyographers with respect to peripheral muscle electromyography. How much more cautious should we be, working in the far more critical field of the extra-ocular muscles!

The present situation of electromyography in clinical diagnosis may be likened to that of tonography in glaucoma. The electromyogram, like the tonogram, is an objective evidence of physiologic processes. As such, it affords a keen insight into basic neurophysiologic mechanisms. In individual cases it provides extremely important information concerning the particular physiology of extraocular muscle. In most instances, however, its findings must be taken with due reserve and in the light of the total picture; it is, then, just another of many diagnostic approaches to muscle problems. Electromyography will not automatically provide diagnoses. It simply illustrates the situation in the final common path, as revealed by the muscle fiber discharge. Just as in peripheral electromyography, its greatest contribution lies in the diagnosis of lower motor neuron and myopathic disorders. I am hopeful, however, that with increasing experience criteria will become available to clearly differentiate the levels of lesions. To distinguish between muscle fiber, nerve trunk, and nuclear and supranuclear lesions would be a most important contribution.

Etiologic diagnoses are difficult, but one does see tendencies for characteristic discharge patterns in a few conditions. It is unlikely that such diagnoses can be made with any certainty in a wide variety of diseases on the basis of electromyographic information alone.

Furthermore, ocular electromyography affords a wire tap on cerebral activity. It is, therefore, par excellence, a tool for the neurophysiologist and is most exciting in its new revelations concerning fundamental brain processes.

The technique of electromyography is eminently practical and has proven devoid of any serious complications. With experience, the minor complications of subconjunctival hemorrhage and exposure keratitis have proven minimal. It should be emphasized that at the present time I do not use a lid speculum, thus cutting down on corneal exposure. With care, artefacts due to lid movement are easily avoided. Furthermore, I use a variety of electrodes of different gauges. The standard 27-gauge needle concentric electrode has proven most useful. I have also used finer-gauge concentric electrodes (up to No. 31). Such needles are extremely light and atraumatic. Their chief difficulty, apart from construction, is a tendency to be displaced during eye movements.

Of particular value in many cases is a skin approach to the inferior oblique muscle. This is the easiest of all muscle insertions. The electrode is inserted through the skin just above the lower orbital margin at its medial aspect. No ocular or skin anesthetic is required. Usually the electrode is placed in the belly of the muscle with one or perhaps two thrusts. There is no problem of corneal exposure or subconjunctival hemorrhage. This has proven of great value in the study of cases of myasthenia gravis in which disturbances of elevation were present. The most difficult insertion is that of the superior oblique, because of its posterior muscle belly. In testing the inferior rectus care is necessary to avoid recording the inferior oblique (this can also be a problem with the lateral rectus). The levator, superior rectus and medial rectus present no problems.

Reliability of Recordings.—Considerable variation in the innervational pattern may be produced by altering the electrode position. However, electrodes placed at different levels within the exposed muscle during a surgical procedure reveal that the general pattern of innervation is much the same at the origin and toward the insertion of the muscle (Fig. 1). It is likely, therefore,

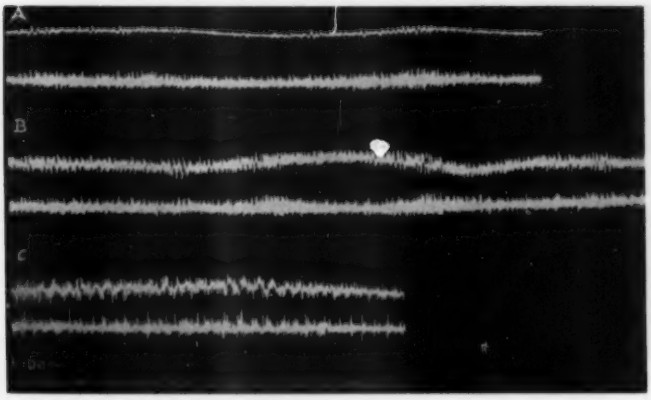


Fig. 1. — Electromyogram (EMG) of left medial rectus under direct surgical exposure. Upper trace, origin of medial rectus; lower trace, near insertion. *A*, *B*, and *C*, slightly different electrode positions, showing similar activity of different units.

that a given electrode position will record the innervational status of the muscle so long as viable fibers are present. However, if one inserts a fine concentric electrode in an atrophic or fibrotic area there will certainly be a loss of innervational pick-up.

Diabetic Neuropathy

Diabetic third nerve lesions are clinically characterized by a sudden onset of paralysis, which in most cases clears rapidly and spontaneously. Denervation, although reported, has not been encountered in my experience, nor does aberrant regeneration usually follow such lesions. Electromyography in a number of such patients showed that the ocular muscles exhibited minute potentials or none at all, the trace being quite silent (Figs. 2 and 3). This is a characteristic of lower motor neuron damage and, in particular, of trunk lesions. Nuclear lesions are less likely to exhibit silence of all the third nerve complex. The appearance is suggestive of tourniquet-type paralyses, in which a conduction block prevents the discharge of muscle fibers. The following are illustrative examples.

A 72-year-old diabetic woman developed a total left third nerve palsy with orbital pain. The eye sat at the outer canthus but could be adducted almost to the midline. Peripheral polyneuritis was present. Very faint activity in the medial rectus was generated on adduction, and no activity was present on abduction (Fig. 2). A tiny remote unit was detected in the levator. No fibrillation was observed. The condition improved over a period of months.

A 60-year-old man developed orbital pain on the right side. One week later an external third nerve ophthalmoplegia developed, with some retention of elevation and

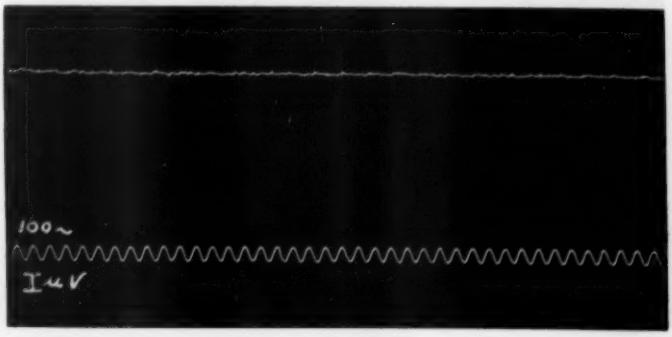


Fig. 2.—EMG of paretic medial rectus. Very tiny activity present.

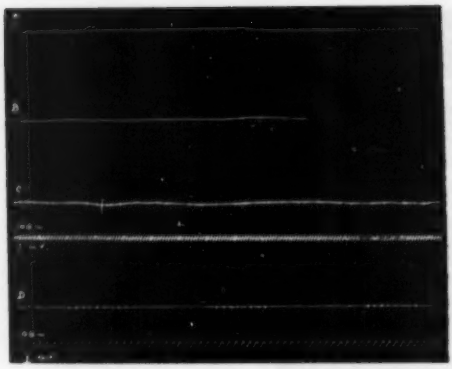


Fig. 3.—EMG of paretic right medial rectus. *A*, abduction, no activity; *B*, slight adduction, very faint activity; *C*, maximal effort at adduction, tiny potentials; *D*, adduction, runs of tiny single units.

depression. This improved in six weeks. A glucose-tolerance test was borderline. Electromyography of the right medial rectus showed slight activity in adduction, with a tiny single unit firing. No activity was present in abduction (Fig. 3). No fibrillation was observed.

In most ocular palsies it is common to encounter potentials which are often of surprising amplitude and frequency. Electrical silence is distinctly unusual. In totally denervating lesions, such as follow cerebral aneurysms or trauma to the trunk of the nerve, there may ensue a period of electrical silence for about 10 days to 2 weeks, after which fibrillations appear. These are the hallmark of denervation. If a suddenly paretic muscle exhibits electrical silence for more than two weeks, one should consider the possibility of diabetes. This may not be a hard and fast etiologic distinction, but it has thus far proven of considerable interest. Such an innervational finding may indicate a focal lesion in the nerve trunk or nucleus which by compressing the nerve fibers or by toxic action causes a conduction block. The subsidence of the pathologic process would then permit resumption of activity. Other compression lesions doubtless occur, as seen in a case of orbital tumor with third-, fourth-, and sixth-nerve palsy (Fig. 4).

Further Studies in Aberrant Regeneration

Many cases have been accumulated of third-nerve lesions followed by the syndrome of aberrant regeneration. These all showed simultaneous activity of the third-nerve complex of muscles. In partial forms the abnormal activity may be limited to a few muscles. Of interest is a case in which the aberrant process primarily involved the lower division of the oculomotor nerve following a fracture of the skull. In this instance, some 10 degrees of elevation was still possible but on attempted depression

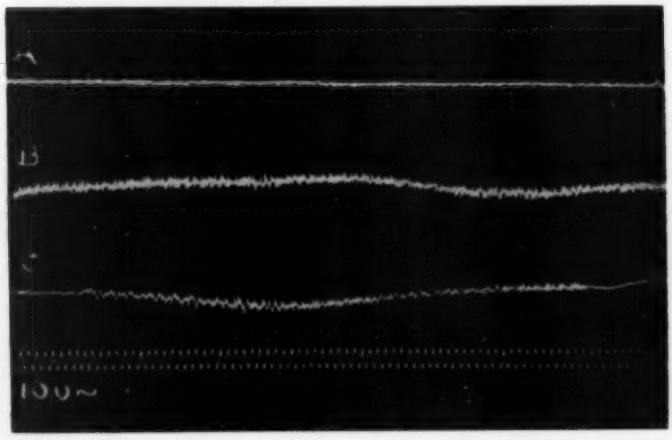


Fig. 4. — EMG of paretic lateral rectus and medial rectus. *A*, lateral rectus, very faint activity; *B* and *C*, medial rectus on adduction, fair activity poorly sustained.

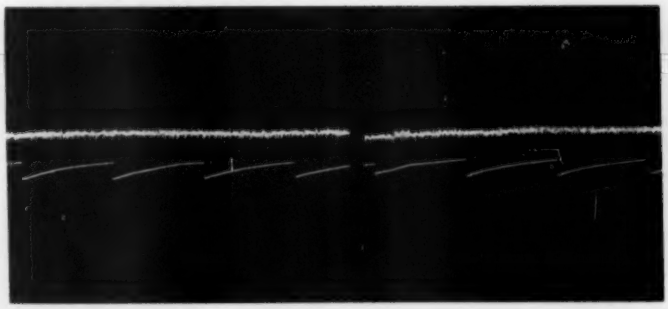


Fig. 5.—EMG of left inferior oblique, integrator below. Aberrant regeneration. Left, gaze up and in; right, gaze down and in; note equal activity.

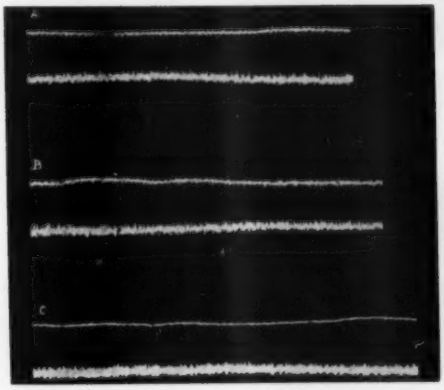


Fig. 6.—EMG of left inferior oblique above, left medial rectus below. Aberrant regeneration. *A*, primary position; *B*, adduction—both increase; *C*, abduction, oblique inhibited while medial rectus increases anomalously.

the globe remained immobile or adducted and the levator did not retract. The pupil constricted on adduction and widened on abduction. Sampling of the left inferior oblique, the left medial rectus, and the left inferior rectus showed that simultaneous discharges occurred in these muscles on attempted adduction, elevation, or depression (Figs. 5, 6, and 7). In another traumatic case, with the patient recumbent, retraction of the globe was noted on attempted elevation. Despite the complete absence of elevation or depression, a full range of horizontal movement was possible, with good fusional amplitudes. Although, in general, the discharge shows reduced voltage, in some instances large motor units are retained (Fig. 8). Anomalous innervation of the medial rectus in abduction has also been noted (Fig. 6). In several cases the vertical muscles showed a marked difference in activity on horizontal movement. Abduction was invariably associated with diminution of activity (Figs. 6 and 9). In this way a horizontally rotated striped drum elicited opticokinetic nystagmus in the inferior oblique.

Further Studies in Myasthenia Gravis and Muscle Dystrophy

Continued experience with the analysis of extraocular muscle potentials in patients with known or suspected myasthenia gravis has been most informative. The pattern

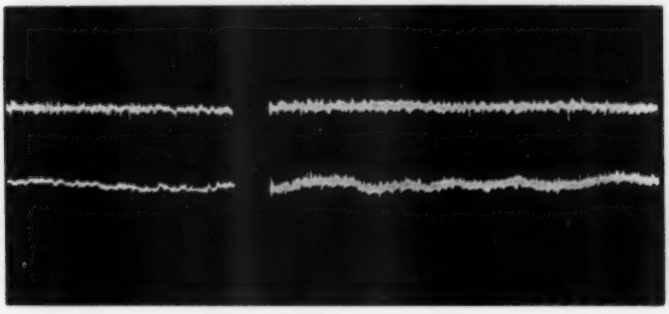


Fig. 7.—EMG of left inferior oblique above, left inferior rectus below. Aberrant regeneration. Left, gaze down; right, gaze up; both are active out of field.

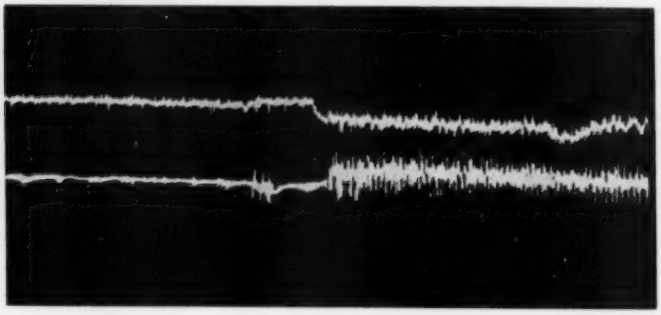


Fig. 8.—EMG of left medial rectus above, left inferior oblique below. Aberrant regeneration. Both recruit from primary position to gaze up.

of motor unit fall-out is so characteristic that the pharmacological test, although confirmatory, may not be absolutely necessary. No other condition has simulated the progressive disappearance of potentials that we have noted in true myasthenic patients. Paretic patterns show nystagmoid recruitment and the maintenance of a basic level of innervation over a period of several minutes. In the myasthenic patient, during attempted gaze in the direction of action of an involved muscle there is a regular fall-out of potentials (Fig. 10). When edrophonium is given there is a prompt return of activity (Fig. 11). I would stress that in numbers of instances the increased activity is not accompanied by much or any improvement in motility. These are the

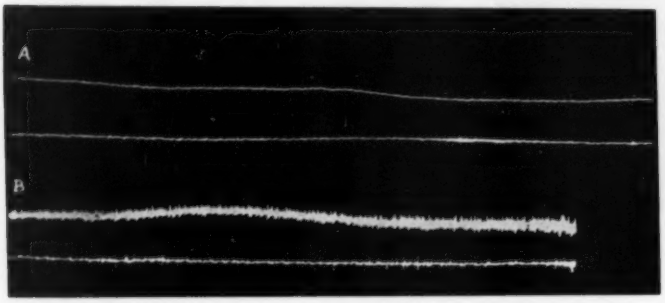


Fig. 9.—EMG of left medial rectus above, inferior oblique below. Aberrant regeneration. *A*, primary position, very little activity; *B*, adduction, both recruit.

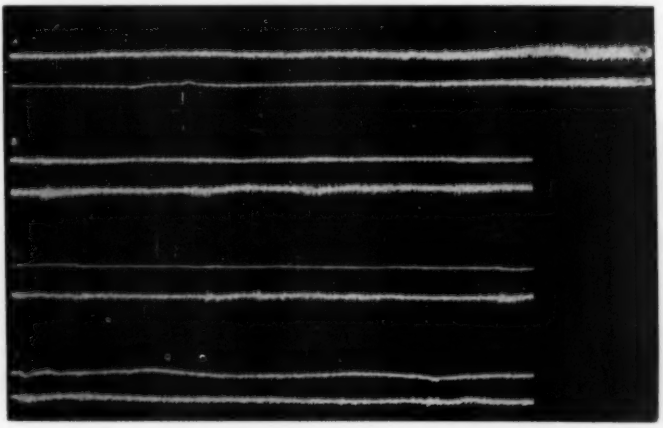


Fig. 10.—EMG in myasthenia. Upper trace, levator; lower trace, inferior oblique. *A*, primary position to gaze up; *B*, gaze up one minute later, fall-off; *C*, gaze up one and a half minutes later, marked fall-off; *D*, recovery after gaze was lowered.

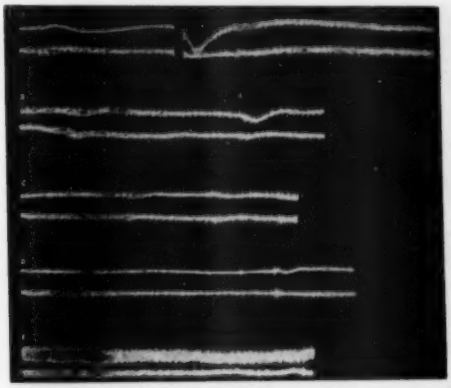


Fig. 11.—EMG in myasthenia. Upper trace, levator; lower trace, inferior oblique. *A*, left, base-line activity; right, 15 seconds after edrophonium (Tensilon), marked increase; *B*, 30 seconds after edrophonium; *C*, 1 minute after edrophonium, fall-out; *D*, 2 minutes later, marked fall-out; *E*, recovery after period of rest.

problem cases in clinical practice and the particular area in which electromyography affords valuable help. Many cases have been referred to our laboratory from an extremely active myasthenic clinic. Our findings have been in close agreement. Furthermore, we have been able to rule out the diagnosis in cases tentatively labeled myasthenia which exhibited dystrophic (Fig. 12), paretic (Fig. 13), or normal electromyographic patterns.

Of particular interest is a form of myopathy or muscle dystrophy in which a limited response to antimyasthenia drugs occur. Osserman classifies these as a dystrophic form of myasthenia; e. g., two sisters with the typical appearance of progressive ocular muscular dystrophy exhibited almost total ocular immobility and ptosis. One showed a slight improvement of muscle function with prostigmine. Her electromyographic pattern of relatively abundant but low-voltage motor units was

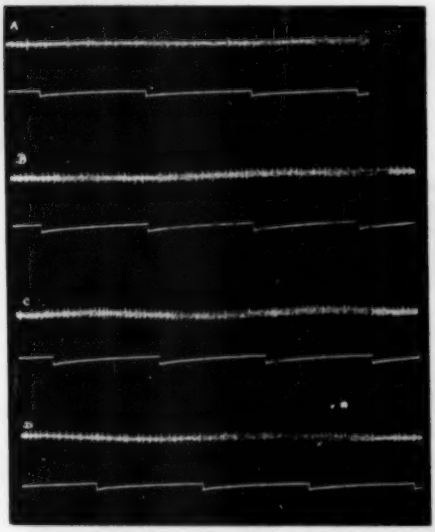


Fig. 12.—EMG in myasthenia-dystrophy. Upper trace, lateral rectus; lower trace, integrator. *A*, base-line activity; *B*, after edrophonium, increases; *C*, two minutes later, fall-out; *D*, two and one-half minutes, marked fall-out.

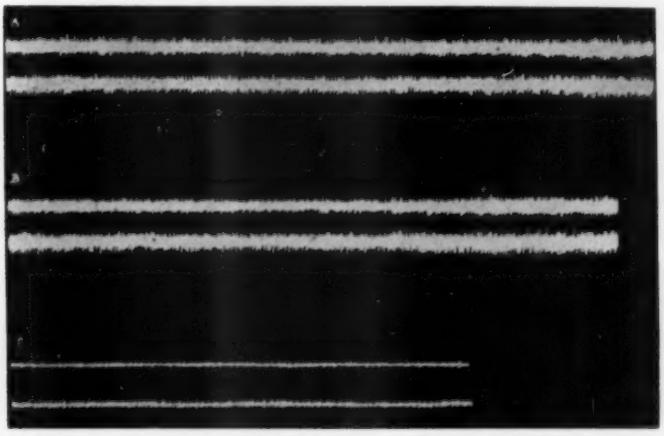


Fig. 13.—EMG in suspected myasthenia. Upper trace, right inferior oblique; lower trace, left inferior oblique. *A*, gaze up, right inferior oblique shows paretic pattern; *B*, gaze up several minutes later, more marked paretic pattern; *C*, primary position after several minutes, low activity in paretic right inferior oblique.

Fig. 14.—EMG in myasthenia-dystrophy. Upper trace, left inferior oblique; lower trace, integrator. *A*, no activity; *B*, after edrophonium, no activity; *C*, left medial rectus, very faint activity.

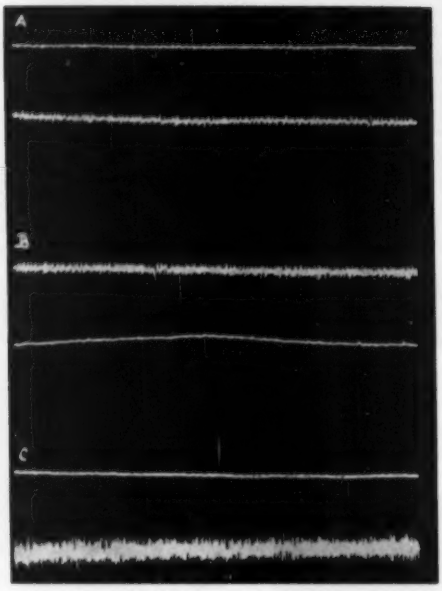


Fig. 15.—EMG of right medial rectus above, right lateral rectus below; dystrophy. *A*, primary position; *B*, adduction, dystrophic pattern of medial rectus; *C*, abduction, normal pattern of lateral rectus.

characteristic of dystrophy but, unlike other such cases, revealed a slight increase of amplitude after edrophonium (Fig. 12). The other sister had practically no recordable potentials in any extraocular muscle and showed no response to edrophonium (Fig. 14).

Also of interest is the occurrence of localized forms of muscle dystrophy, showing the characteristic low voltage, with abundant discharge in only one or two of the extraocular muscles (Fig. 15). I have observed involvement of the medial rectus or levator alone, with functional impairment limited to these muscles. Such localized forms accord with the spotty distribution of this disease in systemic musculature. It is possible that this may represent an early stage in the evolution of the disease, but symmetrical involvement is the rule in the generalized type. In general, there is little or no value in the treatment of dystrophies with the antimyasthenia drugs.

Dysthyroid Ophthalmoplegia

The palsies associated with thyroid exophthalmos have been referred to as myopathies because of the appearance of discrete pathologic changes of the muscle tissue. A study of 12 such palsies revealed that in only 1 equivocal instance did the innervational pattern of myopathy appear. On the other hand, neurogenic palsy patterns occurred in most. The early case, despite the paresis, reveals a normal innervational pattern (Fig. 16). It is likely that the turgescence of the orbital tissues in thyroid exophthalmos does impose some mechanical impediment to motility. Al-

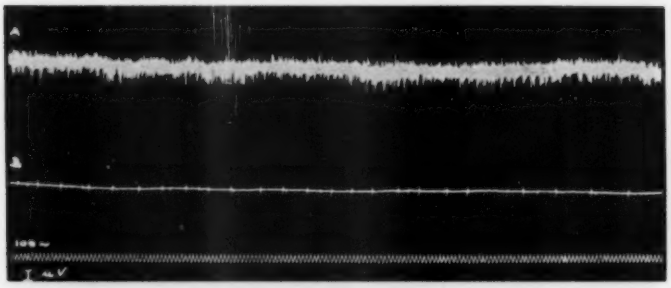


Fig. 16. — EMG in exophthalmos. *A*, levator, large amplitude, gaze up; *B*, single unit in gaze down.

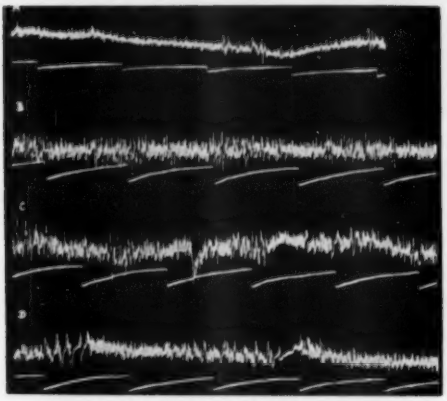


Fig. 17.—EMG in exophthalmos. Upper trace, right superior rectus; lower trace, integrator. *A*, primary position; *B*, gaze up; *C*, irregularity becoming marked; *D*, irregularity very marked.

though the muscles fire normally, they seem unable to move the globe to any extent. Later in the disease definite paretic patterns of innervation (Figs. 17 and 18) appear, which, in the more congestive forms, become quite marked (Fig. 19). The partially myopathic pattern cited was found in the medial rectus of a person with thyrotoxic exophthalmos. Adduction and elevation could not be accomplished. The medial rectus exhibited a low-voltage, fairly abundant, but irregular discharge. The inferior oblique showed a neurogenic paretic pattern (Fig. 20). It therefore seems more appropriate to consider the severer disorder as a neurogenic palsy based either upon damage to the nerves through orbital turgescence or upon specific toxic factors affecting the nerve fibers. It is probable that some loss of muscle fiber response occurs as well, but this is a minimal part of the innervational picture. In a healthy euthyroid young male with marked monocular exophthalmos of unknown etiology no limitation of motility was found and the electromyographic pattern of the recti and

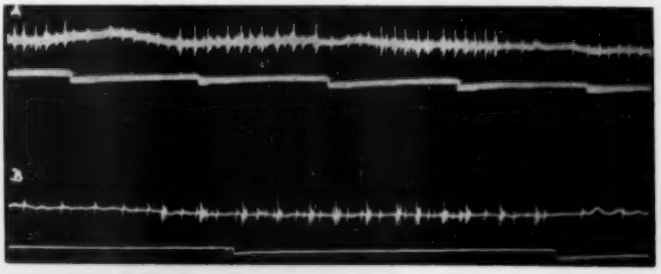


Fig. 18.—EMG in exophthalmos. Upper trace, right superior rectus; lower trace, integrator. *A*, polyphasic and single units; *B*, polyphasic and single units at faster speed.

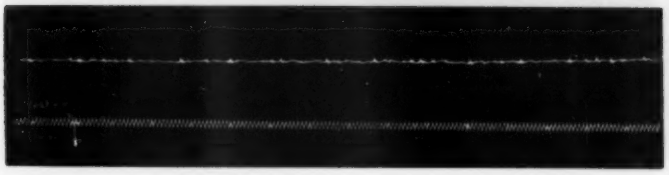


Fig. 19.—EMG in exophthalmos. Lateral rectus exhibiting low-amplitude single unit (possible fibrillation).

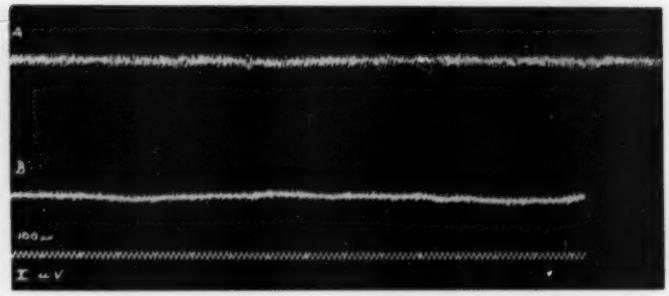


Fig. 20.—EMG in exophthalmos. *A*, left inferior oblique, gaze up, diminished activity; *B*, left medial rectus, gaze in, low amplitude.

oblique was completely normal. Exophthalmos, per se, therefore, does not seem responsible for the pareses. Muscle palsies have been encountered most frequently in congestive forms of thyrogenic exophthalmos, but they have also been seen in apparently noncongestive types. The latter exhibited innervational patterns differing little from the normal.

Lid Lag.—The lid lag, so characteristic of thyrotoxic exophthalmos, does not reveal an abnormal discharge pattern in the levator muscle. The inhibition of activity upon looking down is indistinguishable from the normal. The failure of the lid to accompany the globe may be ascribed to mechanical factors, comparable to the paretic phenomena in some of these cases. Of course, an autonomic factor may play some part, but in all probability this is not a major feature.

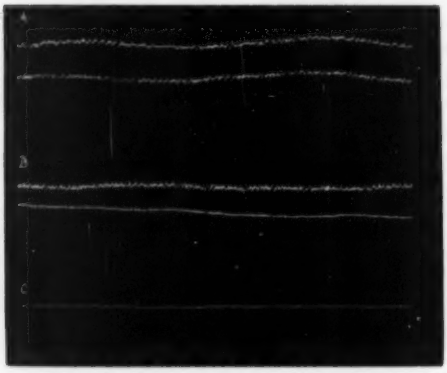


Fig. 21.—EMG in lid retraction. Upper trace, right levator; lower trace, left levator. *A*, gaze up; *B*, primary position, right levator very active; *C*, gaze down, both inhibited, the left completely.

Lid Retraction.—Lid retraction was studied in a patient with slight hypothyroidism. The right palpebral fissure was considerably wider than the left in all directions of gaze, and the innervational activity of the right levator was also greater than the left in the primary position and on looking down (Fig. 21). A retrobulbar injection of pro-

Fig. 22.—EMG in lid retraction. Upper trace, levator; lower trace, integrator. *A*, gaze up; *B* and *C*, gaze down; *D*, gaze up to down, with fluctuating innervation.

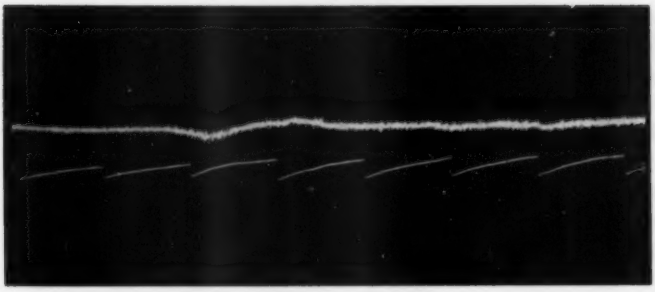


Fig. 24.—EMG in tendon sheath syndrome. Upper trace, inferior oblique; lower trace, integrator. Primary position to gaze up, good activity.

caine in the right orbit abolished activity of the levator and reduced the width of the palpebral fissure to a few millimeters.

In another patient with exophthalmos the lid was retracted when the gaze was lowered. The levator revealed a fluctuating level of innervation before complete inhibition was attained (Fig. 22*).

Widening of the palpebral aperture on abduction of the eye in a patient with a paretic lateral rectus (seen also in the retraction syndrome) is also accompanied by increased innervation to the levator.

Superior Oblique Tendon Sheath Syndrome (Harold Brown)

A 6-year-old child and an adult exhibiting the typical features of this syndrome were studied. The affected eye could not elevate above the midline in adduction but elevated well in abduction; the movement described a sliding plane. No overaction of the superior oblique was present, and no deviation was present in the primary position. Both exhibited V-type exotropia in upward gaze. In both cases the inferior oblique fired entirely normally, which makes it inescapable that the failure to elevate in adduction must have been due to mechanical reasons (Figs. 24 and 25). The adult showed comparable firing in the inferior obliques of both sides. He also exhibited an increase of firing of the lateral rectus in upward gaze. This will be described in a study of "A"- and "V"-syndromes. The fact that cutting the superior oblique tendon sheath does not result in improvement in many of these cases is presently a mystery. Presumably other structural changes may be present.

* Figure 23 deleted on proof.

Moebius Syndrome

An infant 1½ years old exhibited a bilateral sixth- , seventh- , and fifth-nerve palsy with total inability to abduct either eye. Slight adduction was possible, and convergence ability was good. No retraction was noted. Muscle balance varied from orthotropia to esotropia. The lids could not be completely closed, and a well-marked Bell's phenomenon was noted. When he attempted to follow an object a convergence movement was substituted for a version—the pupils also constricting. No response to vestibular stimulation was elicited, and no blink reflexes were obtained. Bilateral corneal anesthesia was present. Vertical movements were unimpaired. The forehead

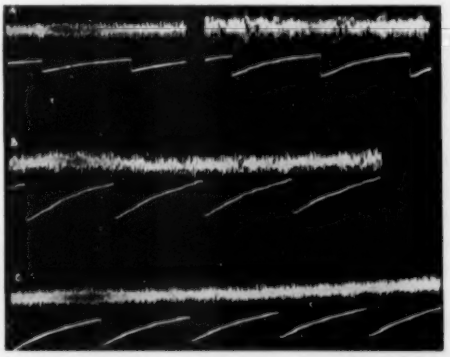


Fig. 25.—EMG in tendon sheath syndrome. Upper trace, inferior oblique; lower trace, integrator. *A*, right inferior oblique; left, gaze out; right, gaze in; *B*, left inferior oblique, gaze in; *C*, left inferior oblique, gaze out; both *B* and *C* equally active, more so in adduction.

was flat and unwrinkled, and the eyes were deep-set. The face showed asymmetry and total absence of expression. The mouth was slack. Multiple congenital anomalies were present, including absence of the left hand and wrist.

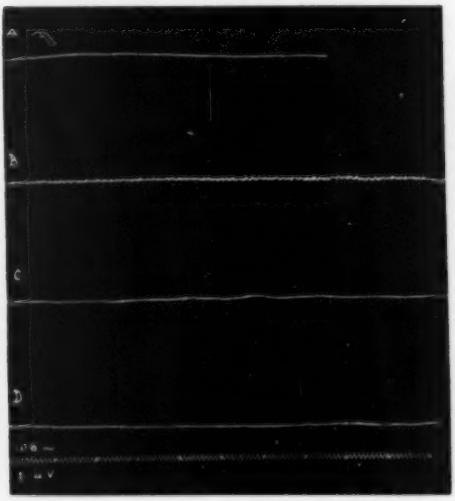
Electromyography of the lateral rectus revealed almost no potentials (Fig. 26). Only one area showed some irregular activity of low amplitude and frequency. A curious gritty sensation was encountered during electrode insertion, suggesting fibrosis.

Electromyography of the orbicularis oculi and orbicularis oris revealed almost complete electrical silence. A few insertion potentials were obtained from the orbicularis oris.

The electromyographic pattern, the multiple nerve involvement, and the evidence of gaze palsy suggest a neurogenic lesion.

This pattern is consistent with a congenital aplasia of the nuclei with consequent absence of innervation and, in addition, a supranuclear involvement. A primary muscle aplasia cannot be ruled out but seems unlikely.

Fig. 26.—EMG in Moebius syndrome. *A*, lateral rectus, silent; *B*, lateral rectus, one area of low irregular activity; *C*, orbicularis oculi, silent; *D*, orbicularis oris, silent.



This case is noteworthy in that it is the youngest subject in whom electromyography has been performed. The child was slightly sedated and mummified. No great difficulty was presented, and good humor was evidenced immediately after the procedure.

Vertically Incomitant Horizontal Strabismus

A great deal of interest has been aroused by studies of so-called A- and V-syndromes, in which comitant esotropia or exotropia increases in the straight up or straight down position. Urist has brought this condition to the attention of American ophthalmologists.[11] He has emphasized the importance of the lateral recti in the upper field and the medial recti in the lower field. Thus, an esotropia increasing in gaze down (V-type) is said to be due to medial recti overaction, whereas an esotropia increasing in gaze up (A-type) is due to lateral recti underaction. Exotropia increasing above (V-type) would be due to overaction of the lateral recti, and exotropia increasing below (A-type) would be due to medial recti underaction. He therefore urges initial horizontal muscle surgery, to obtain vertical comitancy.

Others claim primacy for the vertical muscles in these variations and recommend vertical muscle surgery as an initial procedure to attain vertical comitancy.

Because of these studies, the straight up and straight down positions should be included in the diagnostic examination of strabismus.

We have carried out electromyographic studies of vertically incomitant horizontal strabismus to determine the role of the horizontal and vertical muscles. It is difficult to evaluate the effect of vertical muscles, since they show only the expected recruitment in their fields of action. Whatever accessory abductory or adductory action they possess is a mechanical consequence of the relation of the muscle plane to the center of rotation. There seems little doubt that such actions do exist.

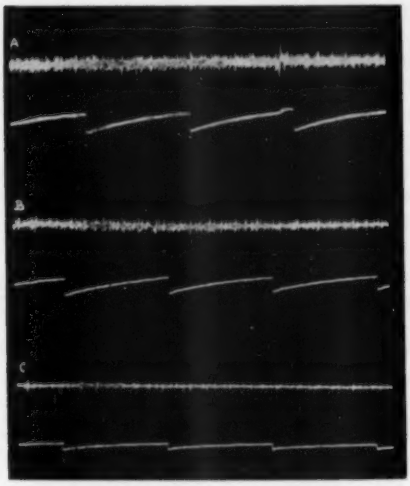


Fig. 27.—EMG in V-type exotropia. Upper trace, right lateral rectus; lower trace, integrator. *A*, gaze up and out; *B*, gaze out; *C*, gaze down and out. Activity greatest in upper gaze.

The horizontal recti, however, in nearly all of the 10 cases of the four types described, have shown innervational changes corresponding to the deviation. Thus, in V-type exotropia the lateral recti increased and the medials were reciprocally inhibited in upward gaze (Fig. 27). In A-type exotropia the identical changes occurred in downward gaze (Fig. 28). Corresponding alterations appear in A- and V-esotropias (Fig. 29).

It seems clear that, at the very least, the horizontal recti must play some part in the varying angle of strabismus. In only a few cases of A- or V-syndromes did the horizontal recti exhibit no particular change in vertical gaze (which is the normal situation in vertically comitant horizontal strabismus). In these cases the vertical incomitancy must be ascribed to the vertical muscles.

From these preliminary observations it would appear wise to surgically attack the horizontal muscles before the vertical ones. Where gross vertical imbalance exists, however, classical rules must still be followed.

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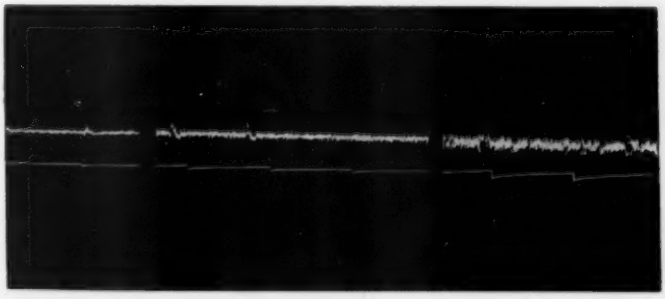

Fig. 28.—EMG in A-type exotropia. Upper trace, left lateral rectus; lower trace, integrator. Left, gaze up; center, primary position; right, gaze down.

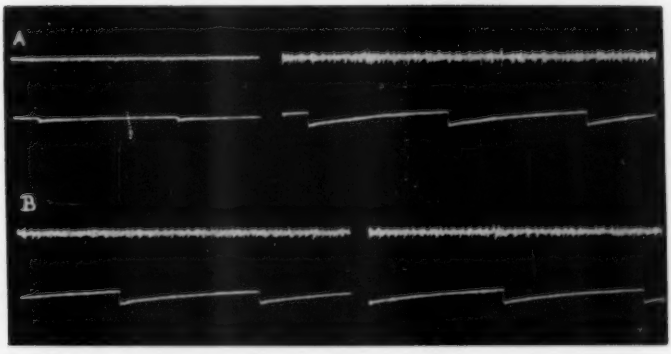

Fig. 29.—EMG in A-type esotropia. Upper trace, right lateral rectus; lower trace, integrator. *A*, left, gaze up, slight activity; right, gaze down, good activity, O. S. fixing. *B*, left, O. D. straight up; right, O. D. straight down. No difference. O. D. fixing.

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Five Years' Experience with the Ridley Operation

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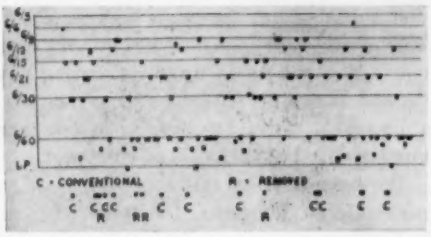
Four years ago, before this Section, we presented a preliminary report on the Ridley operation. Today we report on 115 cases over a five-year period. Although the present account gives the data on six times as many cases as the preliminary one, we realize that it is by no means conclusive and that it will require many more years before this operation and its modifications can be properly evaluated. Unfortunately, in this country, few mature, experienced ophthalmic surgeons have attempted the Ridley operation, and so there is practically no literature on it and the various modifications that have been proposed. We believe that we have had more success with this operation than others. Barraquer [1] has remarked this, and the committee appointed by the American Academy of Ophthalmology and Oto-Laryngology was evidently favorably impressed after viewing a number of our patients. This success, we believe, may be due to (1) a greater number of cases, and therefore more experience; (2) selection of patients; (3) a carefully worked out technique, and (4) teamwork.

When a patient consults us about a Ridley operation we discuss the situation frankly, explaining that it is mainly indicated in cases of unilateral cataract, so that after operation both eyes can be used together; that it is more hazardous than the conventional operation, but in competent hands not unduly so, and that should the other eye become cataractous it would also require a Ridley operation. We discourage the operation in persons with bilateral cataracts and warn all patients that certain things may occur at time of operation necessitating conversion to a conventional cataract extraction. We say that the objection that the acrylic lens will act as a foreign body has not been borne out by our experience.



Graph 1.—Thirty-nine per cent of patients have vision of 6/21 or better without glasses. Ten per cent of the operations were begun as Ridley operations and completed as conventional ones.

Eleven cases were started as Ridley operations but were converted into conventional extractions because of loss of vitreous (six cases), overdilatation of pupil (two cases), dense capsule (two cases), and, in a young girl, a small lens nucleus which could not be removed (1 case). All of these now have good vision with aphakic corrections. Graph 1 shows this and gives visual results without glasses.

We described our technique in the preliminary report. It is designed for simplicity, speed, control of hemorrhage, and minimum trauma. It is based on the belief that in any cataract operation the more quickly the eye is closed and bandaged the less likelihood there is of complications and undue reaction.

The postoperative care is most important and at times difficult. The eye is inspected the morning following operation and 0.5% hydrocortisone and a mydriatic are instilled, the strength of the latter depending on the size of the pupil, its mobility as determined preoperatively, and the degree of reaction. Usually 1% or 2% atropine is used. The eye is dressed again that afternoon and thereafter once daily with similar treatment. We also use prednisone four times daily, decreasing it during convalescence. If, at the end of several days, the pupil is small

and bound down, 1% atropine hot compresses and 0.5% hydrocortisone are ordered every three hours, and, should it be necessary, a neo-synephrine pack is placed in the lower cul-de-sac. The patient is given the use of one eye on the third or fourth day and dark glasses a day or so later. As the reaction subsides and the pupil widens, 2% or 4% homatropine is substituted for the atropine. The patient is usually discharged at the end of two weeks (unless he comes from a distance), with 0.5% hydrocortisone three times a day, but seldom a mydriatic.

We have had several cases with poor results due to improper follow-up. In one instance, a physician who was opposed to the Ridley operation declined to see the patient further.

The patients' ages ranged from 12 to 80 years. They include a surgeon, a commercial pilot, and a locomotive engineer. We doubt if these men could have resumed their duties without the Ridley operation. Indeed, the commercial pilot astounded the Civil Aeronautic Examiners with his remarkable depth perception. Also included are four diabetics and two patients with uveitis, all of whom did well.

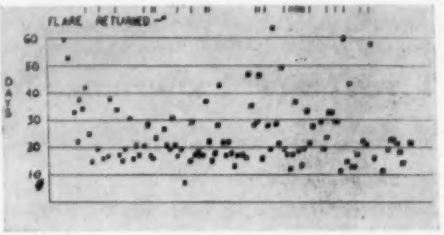
About 30% of our cases have had striped keratitis which cleared up rapidly.

Four cases had prolapsed iris; in all the prolapsed portion was small. In two of these it was necessary to use scissors to enlarge the incision, and we believe that this may have been a causative factor.

Some cases showed phakodonesis postoperatively, but this soon disappeared.

There were five cases of anterior chamber hemorrhage. Four of these were spontaneous, slight, and cleared promptly. One patient rubbed his eye, and the resulting hemorrhage required 16 days to absorb. Another had a hemorrhage six weeks postoperatively, and, although it was fairly profuse, it cleared promptly, we were told.

We have had to remove four acrylic lenses. With one exception, all of these eyes now have good vision with an aphakic correction. In not one case was the lens dislocated into the vitreous.



Graph 2.—The maximum time after surgery that initial postoperative flare was not found. (Many patients were followed by their local physicians, and several weeks, or months, elapsed between the time that flare was found and the time that it had gone.) Twenty-three per cent had recurrence of flare at a later date.

Five per cent of the cases developed glaucoma, not present in the unoperated-on eye. All of these were controlled, and three were apparently cured. One may have been a case of primary glaucoma.

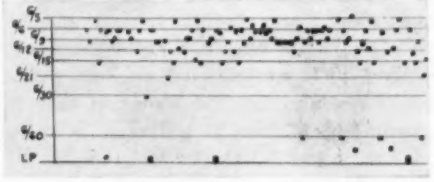
Five per cent had an exudative pupillary membrane that required corelysis, or as Ridley designates it, corepraxia. This consists of severing the exudative membrane covering the pupil with a knife needle. It is not done until the eye is fully quiet.

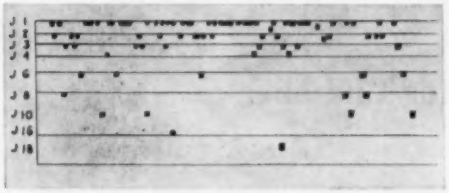
Accurate observation of flare disappearance and recurrence in most cases was difficult, as the great majority of patients were followed by their local ophthalmologist. Graph 2 shows this.

Traumatic cataracts, and cataracts in young persons, were more difficult to manage and did not offer as good prognosis, the former because of concomitant injuries to structures, in addition to the lens, and the latter owing to difficulty in managing the pupil and getting out all lens material completely.

Graph 3 shows our visual results with glasses. In our opinion, direct vision is

Graph 3.—There are 74% of patients with vision of 6/12 or better with correction; 64% have vision of 6/9 or better with correction.





Graph 4.—Near vision with glasses; 60% have J-3 or better.

better in the modern conventional cases, but field of vision, orientation, and perspective are better in the Ridley cases. This is in accord with what one would expect from such a change in an optical instrument. Ridley[2] expresses it when he ascribes the better direct vision of conventional cases to magnification of the retinal image.

Most cases are myopic postoperatively, with a cylinder of around 90 degrees. For this reason we have been averse to operating on myopes, but our experience with five cases has shown no material increase in the myopia of the operated-on eye.

Many patients show remarkably good distant and, at the same time, near vision without glasses. Most patients can read rather well without glasses. Graph 4 shows near vision with glasses.

Cooper and Patel[3] suggest that since acrylic is soluble in alcohol possible intolerance in chronic alcoholics should be watched for. Curiously enough, two of our worst cases were alcoholics; one developed iris bombé, and another, a dense pupillary exudative membrane.

Probably the greatest danger in the Ridley operation is the postoperative reaction. This is not surprising if one considers the operation as two procedures, namely, a cataract extraction and a lens implantation. The reaction can be so severe as to result in iris bombé and practically destroy the eye. Fortunately, we have had only one such case, an alcoholic, referred to previously. Others have had less severe reaction, with hypopyon, and we have had four such cases. This was at first very frightening, but we found that the hypopyon disappeared in a day or so, and all of these eyes made a good recovery. Moderate reaction is fortuitous, as it creates firm adhesions that hold the acrylic lens in position. This postoperative reaction is evidently due to the implantation and probably stems from iris manipulation, and possibly anterior chamber irrigation; so these should be kept at a minimum. We believe it is due also to individual susceptibility, since in the bilateral cases that we have done it has been about the same in each eye. If this reaction could be foreseen or forestalled, most of the hazard of the operation would disappear. All cases have a certain amount of exudation into the anterior chamber and redistribution of pigment that frequently results in heterochromia.

In conclusion, we hope that this paper will be viewed as a sincere attempt to evaluate a daring and dramatic replacement which is so simple in concept that, like many other great discoveries, one wonders why it was not thought of earlier. If it, or any of its modifications, prove feasible and practicable—and we believe they will—its benefits can hardly be calculated. Eyes now considered lost from an industrial standpoint will be saved, and patients engaged in work demanding binocular vision will have their livelihood restored. Who knows but what in days to come some ingenious surgeon may not only replace the cataractous lens but may devise some means of changing its focus or making it bifocal, so that from an ocular standpoint it will really result in rejuvenation.

2118 Locust St. (3).

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Acrylic Lenses in the Anterior Chamber

Experimental Studies in Animal Eyes

J. H. KING JR., M.D., Washington, D.C., and LIEUT. COL. R. A. SKEEHAN JR. (MC), U.S. Army

The correction of certain refractive errors, either intrinsic or resulting from removal of the lens, by insertion of a plastic lenticulus within the eye has been attempted by several ophthalmic surgeons. The first to report the use of intraocular acrylic lenses after monocular cataract extractions was Ridley,[1] in 1952. His purpose was to overcome the disadvantages of correcting the aphakic eye by spectacles, which include magnification of the image, distortion and displacement of images outside the optic center, and marked limitation of the fields of vision. He obtained binocular vision by inserting an intraocular lens behind the iris (Fig. 1*A*). The lens he used was made of polymethyl methacrylate, fashioned slightly smaller than the human lens. It was 8.35 mm. in diameter and 2.4 mm. in thickness; the radius of curvature of the anterior surface was approximately 9 mm., and that of the posterior surface was 5.3 mm. Its refractive index was 1.33, and its refractive power in aqueous, 24 D. A groove around the equator made it possible to hold the lens by special forceps. He introduced the acrylic lenticulus behind the iris, usually immediately after extracapsular cataract extraction, although he employed the intracapsular technique in some cases.

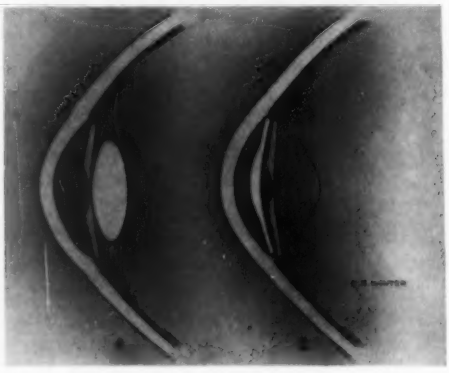
Ridley stated that the lens was well tolerated and that the mild plastic iritis it induced subsided within a few weeks. The operation itself proceeded uneventfully in 90% of his cases, and neither sympathetic ophthalmia nor prolonged iridocyclitis occurred subsequently. Most of his patients regained excellent visual acuity and binocular vision, although a few required a low-power spectacle lens.

The Ridley lens has not been widely adopted, chiefly because of the technical difficulty of inserting the lens behind the iris and the increased hazard of postoperative complications that the procedure entails. But the idea of an intraocular artificial lenticulus to alleviate the disadvantages of aphakia and to correct high refractive errors in patients who retain the crystalline lens has stimulated a number of efforts to improve upon the Ridley lens.

Strampelli,[2] Bietti,[3] Apollonio,[4] and others have constructed acrylic lenses which are introduced into the anterior chamber. The shape of the lens varies with each surgeon, but essentially it consists of a plastic strip with a central lenticulus containing the refractive component (Fig. 1*B*). The length of these lenses varies from 11

From Georgetown University Medical Center (Dr. King); from the Ophthalmic Pathology Section, Armed Forces Institute of Pathology (Lieut. Col. Skeehan).

Fig. 1.—Schematic drawing of placement of acrylic lenses. *A*, Ridley lens posterior to iris. Posterior capsule of crystalline lens is intact. *B*, Strampelli lens in anterior chamber. Tips of lens are in contact with corneoscleral trabeculae.



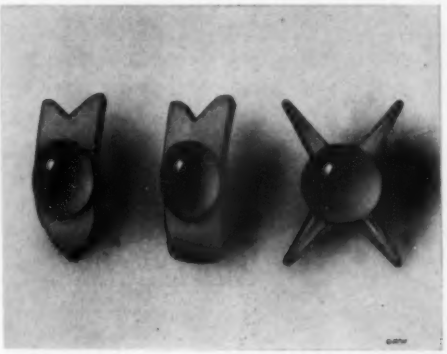


Fig. 2.—Acrylic lenses for insertion into the anterior chamber of the eye. The length of each may vary between 11 and 12 mm. *A*, Strampelli's modified lens, 5 mm. wide. *B*, Bietti's lens, 6 mm. wide. *C*, Bietti's original lens.

to 13 mm., depending upon the diameter of the anterior chamber. They are constructed so that the refracting surface is centered over the pupil, while the extremities rest in the anterior chamber angle to act as supports. Strampelli's lens (Fig. 2*A*) is in the form of a meniscus with a radius of 12 mm., the average curvature of the sclera. Its shape is somewhat rectangular, 4.5 mm. in width and 12 mm. in length. In one end is a central V-shaped notch to facilitate peripheral iridectomy, and the other end is rounded. Bietti's lens (Fig. 2*B*) is similar but slightly wider, 6 mm. in diameter, in order to cover a larger pupillary area. His original lens (Fig. 2*C*) had four supporting legs; these, however, did not prevent rotation and permitted too much contact with the chamber angle. The central refracting surface of all these lenses is ground as a plus-minus sphere to correct the refractive error determined by examination of the patient. The contour is such that when the lens is introduced into the anterior chamber its tips rest in the inferior and superior iridocorneal angles, while the slight forward curvature prevents any contact with the iris or posterior corneal surface (Fig. 1*B*). In addition, the posterior surface of the inferior support of the lens is ground with a slight concavity to minimize contact with the iris.

The authors cited are gratified with their results, for the lens has been well tolerated by the limited number of patients on whom it has been used. According to Bietti,[3] the only change apparent in the angle on gonioscopic examination has been a circumscribed area of thickening at the points of contact with the tips of the artificial lens. All authors agree that the advantages of the anterior chamber lens outweigh by far those of the posterior chamber (Ridley) lens. Among these advantages are (1) introduction of the lens into the anterior chamber at a second operation when the eye is quiet, preferably six months or more after cataract extraction; (2) limited deposition of pigment on the lens; (3) better vision for the patient, for each lens is ground to correct the particular refractive error of the individual person; (4) provision for the performance of subsequent discission, if necessary, with the lens in place, and (5) easy removal of the lens if it is not well tolerated. Other than its use in unilateral aphakia, the anterior chamber lens may also be employed for the correction of high refractive errors and anisometropia in an eye still possessing its crystalline lens.

In an attempt to determine the tolerance of the ocular tissues to the plastic material and the possible damage it may inflict on the corneal endothelium, iris, and angle, we began some experiments on animal eyes late in 1954. At that time no report of placing such a lens in the eye of an animal had been published, but since then, Fedrizzi[5] and de Poli and Romagnoli[6] have reported two series in which they investigated the tolerance of rabbit eyes to the acrylic lens in the anterior chamber. In our experiments we inserted methacrylate lens of the Strampelli type (Fig. 2*A*) into the anterior chamber of the eyes of three cats and of three monkeys. We did not extract the crystalline lenses from these eyes, for our interest was centered on the reaction of the intraocular tissues to the plastic material.

The lenses were introduced through a keratome incision at the superior corneo-

scleral limbus, as recommended by Strampelli.[2] The V-notched lens tip was held by smooth forceps, and the rounded end was made to rest in the inferior angle of the anterior chamber. The V-notched end was then teased into the superior angle so that the lens rested in a vertical position. The only difficulty with the operation was in the eye of one cat: the iris diaphragm bulged forward, and the lower end of the plastic lenticulus became entangled in the iris and drew down the pupillary border. In this animal severe iritis persisted for several weeks and subsequently pigment was deposited on the acrylic strip. The

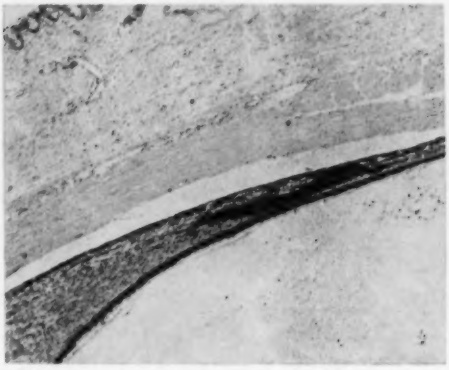


Fig. 4.—Monkey eye. Focal aggregation of lymphocytes in pars plana immediately posterior to the field shown in Figure 3. (A. F. I. P. 574619.)

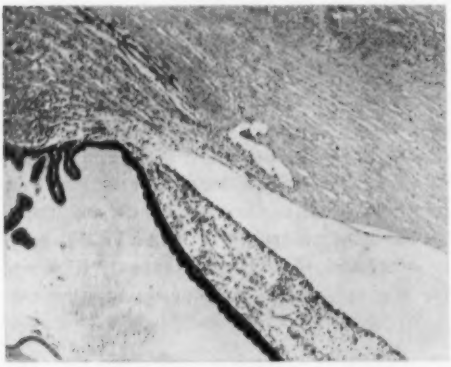


Fig. 3.—Monkey eye. Mild diffuse infiltration of iris and ciliary body by chronic inflammatory cells. Inferior tip of acrylic lens had rested in angle. The plastic dissolved during processing. (A. F. I. P. 57 4618.)

immediate postoperative course in the five other animals was uneventful, with only minimal signs of iridocyclitis for several days. No postoperative activity was noted during 18 months of observation. Two of the acrylic lenses rotated 15 degrees from the vertical axis within two weeks after they were inserted. The lenses in two of the monkey eyes rotated slowly from the vertical axis during the first four postoperative months. There was no external evidence to suggest that movement of the lens incited ocular inflammation.

At the end of 18 months the eyes containing the acrylic lens in each of two monkeys and the one cat in which operative complications occurred were enucleated for histopathological study.

The cornea of the first monkey eye was hazy. Transillumination outlined a round pupil. The eye was opened vertically. The plastic lenticulus was found to have rotated 45 degrees from the vertical meridian in a counterclockwise direction. No gross inflammatory reaction was noted. Microscopically, the iris and ciliary body were slightly but diffusely infiltrated with lymphocytes and plasma cells (Fig. 3). On the side of the corneal section a dense aggregation of lymphocytes was seen in the pars plana (Fig. 4). There was perivascular cuffing

Fig. 5.—Cat eye in which iris was displaced by plastic lens. Chronic iridocyclitis, edema of ciliary body, mild keratitis, perivascular cuffing of limbal vessels, and an organizing fibrovascular membrane resulted from misplacement of the acrylic lens. (A. F. I. P. 574616.)

of the episcleral vessels by lymphocytes.

The cornea of the eye of the second monkey (No. 1070) was clear. Again, the lens had deviated from the vertical, about 30 degrees clockwise. A small streak of pigment lay on the anterior capsule of the lens. There was a small synechia at 12 o'clock. Microscopically slight but diffuse infiltration by chronic inflammatory cells, similar to that in the first eye examined, was seen in the iris but not in the ciliary body.

In the cat eye, chronic inflammatory cells were present in the edematous ciliary body on one side (Fig. 5). Lymphocytic cuffing of vessels was noted in the peripheral cornea. The iris was diffusely infiltrated with lymphocytes, and plasma cells extended into the anterior chamber.

Comment

It would appear that the insertion of an acrylic lens into the anterior chamber produces minimal ocular complication. Fedrizzi [5] found that if the crystalline lens of the rabbit eye was completely absorbed before the acrylic lens was introduced into the anterior chamber no reactive process resulted. However, if cortical material remained in the eye when the artificial lens was introduced, it provoked reactive iridocyclitis. De Poli and Romagnoli [6] introduced the acrylic lens into the anterior chamber of the rabbit eye and also placed pieces of the same material under the palpebral conjunctiva. There was no inflammatory reaction. Histologic studies were not presented.

In our study, when the operation was performed without complication, the postoperative reaction was minimal and not severer than that which follows paracentesis alone. Observation for 18 months revealed no untoward clinical reactions. Microscopically, in the two monkey eyes the chronic reaction was minimal except near the site of the corneal incision. The plane in which the histologic sections were cut included the areas where the acrylic lens touched the iris. The lens itself had dissolved during preparation of the slides. The organizing membrane in the cat eye was very like that seen after intraocular surgery of many types. The presence of the plastic cannot be regarded as the sole cause of the rather severe clinical reaction in the one eye in which the operative procedure was complicated.

Lieut. Col. Joel N. McNair, now Chief, Eye Section, Madigan Army Hospital, Tacoma, Wash., assisted Dr. King at surgery and in the immediate postoperative observation of the animal subjects.

1746 K St. N. W. Wash. D. C.

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Three Cases of Epithelial Invasion of the Anterior Chamber Treated Surgically

JOHN C. LONG, M.D., and GEORGE S. TYNER, M. D., Denver

Epithelial invasion of the anterior chamber was first recognized by Collins and Cross,[1] in 1892, and has since been adequately described and classified by Perera,[2] Calhoun,[3] Pincus,[4] Maumenee and Shannon,[5,6] and others. It is an uncommon but extremely serious complication of perforating wound of the eye or intraocular surgery. Untreated, it may lead to serious visual impairment or loss of the globe. Figures 1, 2, and 3, taken from pathological specimens in the University of Colorado Ophthalmic Pathology Laboratory, illustrate the epithelial downgrowth type of epithelial invasion of the anterior chamber.

These sections demonstrate the mechanism of invasion through the corneal wound into the anterior chamber. The posterior corneal surface, the angle of the anterior chamber, the anterior surface of the iris, and the vitreous face become lined with squamous epithelium. These eyes were enucleated because of intractible glaucoma as a result of obstruction of the chamber angle.

From the Division of Ophthalmology, Department of Surgery, University of Colorado School of Medicine.

It is thought that epithelization occurs following cataract surgery when there is some interference with closure of the corneal wound. Sutures placed too deeply, poor wound coaptation, or the incarceration of iris, vitreous, or lens capsule may be precipitating factors. Early clinical indications of beginning epithelization are persistent iritis or a wound fistula with hypotony.

Later signs are a translucent membrane seen on the posterior surface of the cornea terminating in a gray line, corneal edema over the involved area, and variable degrees of neovascularization. Epithelization of the iris surface may give rise to a masking of iris details and some loss of mobility. Intractible glaucoma ultimately develops.

Fig. 1.—Invasion by surface epithelium through cataract wound, with epithelization of anterior chamber.

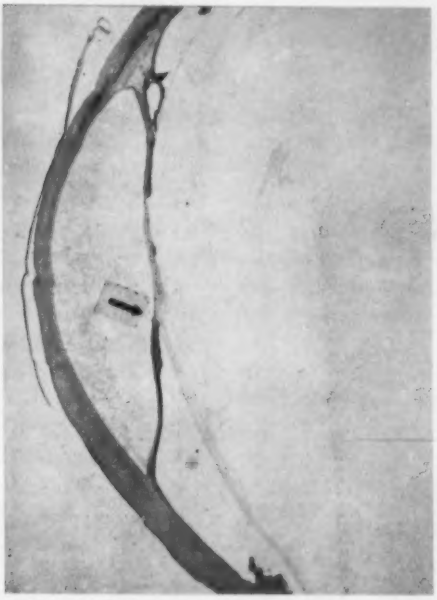


Fig. 2.—Obliteration of chamber angle, with extension of epithelial membrane over pupillary space.

No completely satisfactory treatment for epithelial downgrowth has yet been devised. Somewhat contradictory results have been reported as to the usefulness of irradiation and surgery.

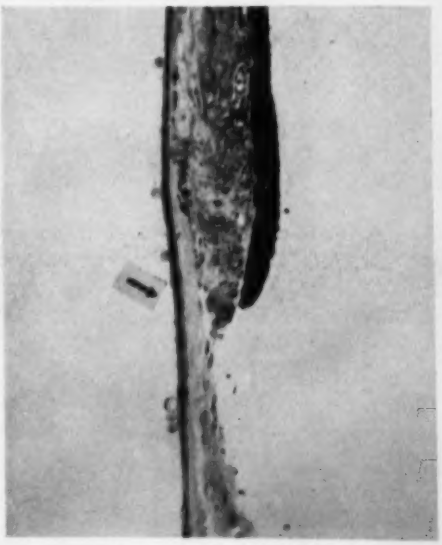
Recently Maumenee[5,7] has reported a surgical technique that seems to offer some advantages in the treatment of this condition. This procedure consists essentially in curettage and removal of the invasive membrane followed by chemical cautery of the involved area. We wish to report three cases so treated.

CASE 1.—An 81-year-old white man underwent an uneventful intracapsular cataract extraction of the right eye on April 10, 1956. A limbus base conjunctival flap was used, with the usual limbal incision. A complete iridectomy was done superiorly. The postoperative course was complicated by a persistent wound fistula and shallow anterior chamber. The referring physician had cauterized the wound lips with diathermy and later resutured the wound. The fluorescein test remained positive after the resuturing.

Examination on May 19, 1956, four weeks after extraction, disclosed a moderately inflamed aphakic eye with a shallow anterior chamber. A fistula was present at the temporal edge of the wound which permitted dilution of fluorescein by escaping aqueous. A translucent membrane with a gray line at its inferior edge could be seen on the posterior surface of the cornea, extending 3 mm. below the upper limbus. The anterior chamber was otherwise not remarkable.

On May 21, 1956, the wound was reopened and the fistulous tract, wound edges, and the upper 3 mm. of the posterior corneal surface were curetted with a chalazion curet. The curetted area was then touched lightly with 70% alcohol on a cotton applicator. After irrigation of the anterior chamber with saline, the wound was resutured. A fornix base conjunctival flap was brought down to the wound and sutured in place. Histological examination of the curetted material disclosed stratified squamous epithelium.

Fig. 3.—Epithelization of anterior surface of iris and vitreous face.



The postoperative reaction was minimal; the anterior chamber reformed promptly, and the fluorescein test became negative. At the last examination, on Jan. 28, 1957, the corrected visual acuity was 20/30. The cornea was opaque over the upper 2 mm. but otherwise clear throughout. There was no inflammation, and the ocular tension was normal.

CASE 2.—A 46-year-old white woman underwent an uneventful round pupil intracapsular cataract extraction of the left eye on June 11, 1956. A limbal keratome and scissors section was made without conjunctival flap. On the seventh postoperative day the eye appeared more hyperemic than usual. On the 11th postoperative day a moderately severe iritis was noted, with some inflammatory exudate at the temporal edge of the pupillary space. This subsided during the next eight days with application of topical atropine and prednisolone. By July 19, 1956, the eye was only moderately hyperemic and the corrected vision was 20/70. On Aug. 17, 1956, the patient was again examined because of recurrent discomfort. At this time there was a grayish translucent membrane extending over the upper one-third of the posterior surface of the cornea. The iris was adherent to the vitreous, and the pupillary space was covered by a membrane. We could not be certain of the nature of the membrane.

On Aug. 21, 1956, the wound was reopened, after a limbus base conjunctival flap had been made. The involved corneal area was curetted with a chalazion curet. A translucent membrane was removed with Arruga forceps. The area was then touched with 70% alcohol. Histological examination of the curetted specimen disclosed stratified squamous epithelium. An inferior radial iridotomy was done.

The postoperative course was stormy, with iritis and anterior chamber exudate. When last examined, on Dec. 28, 1956, the treated corneal area was opaque, the eye was moderately hyperemic, and the pupillary space was occluded by a membrane. The ocular tension was 14 mm. Hg (Schiøtz). The visual acuity was reduced to hand movements.

CASE 3.—A 72-year-old white man underwent an uneventful intracapsular cataract extraction of the right eye on March 21, 1956. A keratome and scissors section was made, with use of a fornix base flap. On April 23, 1956, the eye showed a faint horizontal line about midway across the posterior surface of the cornea. By May 1, 1956, the gray line had descended about 1 mm. further. Corrected visual acuity was 20/40.

The patient was first seen by us in consultation on May 4, 1956. The eye was moderately inflamed, with a grayish membrane extending over the posterior corneal surface and covering slightly more than the upper half of the cornea. Except for a superior iridectomy, the anterior chamber was not remarkable. The fellow eye was normal except for an incipient senile cataract.

Although an epithelial downgrowth was suspected, the diagnosis was not established, and we temporized until July 2, 1956. At that time the appearance of the eye was essentially the same except that a filtering bleb was seen at the upper limbus.

On this date, the wound was reopened without a conjunctival flap. At the site of the filtering bleb a small iris incarceration was noted and excised. A gray membrane extended along the posterior surface of the cornea from the area of iris incarceration. The membrane further extended over the nasal anterior chamber angle, the anterior surface of the iris, and the vitreous face. The membrane was loosened with a curet and grasped with an Arruga forceps. It was removed piecemeal from the cornea, iris, and vitreous face. The cornea, wound edge, and fistulous tract were touched with 70% alcohol. The iris root was dialyzed nasally with a spatula, and radial iridotomy was done inferiorly. The wound was resutured with nonabsorbable surgical (silk) sutures.

Fig. 4.—Sheet of epithelium removed from anterior chamber by curet; × 50.

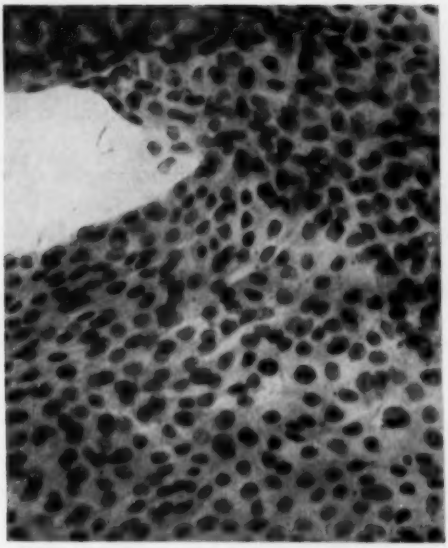


Fig. 5.—Sheet of epithelium removed from anterior chamber by curet; × 500.

Histologic examination of the curetted material and membrane disclosed stratified squamous epithelium (Figs. 4 and 5).

The postoperative course was stormy. During the next 30 days the patient developed keratic precipitates and flare in the unoperated-on fellow eye. A clinical diagnosis of sympathetic ophthalmia was made, and the right eye was enucleated on Sept. 1, 1956. Examination of the enucleated specimen substantiated the diagnosis of sympathetic ophthalmia. The remaining, unoperated-on, eye has improved on orally administered prednisolone. At the present time it is free of inflammation, with a vision of 20/70.

Histologic examination of enucleated eye showed no retained epithelial membrane. The endothelium was lacking over much of the cornea. The iris was fixed to the cornea by broad adhesions. The choroid was packed with lymphocytes and islands of epitheloid cells. This cellular infiltration spared the retina and choriocapillaris but surrounded the emissary vessels. Occasional epitheloid cells were found in the iris and ciliary body.

Summary

Three patients treated for epithelization (epithelial downgrowth) of the anterior chamber following apparently uneventful intracapsular cataract extractions are reported. In one case the cataract section was made with a limbus base flap; in the second, with no flap, and in the third, with a fornix base flap. In two cases a wound fistula was demonstrated. Incarcerated iris was present in one of the fistulas. In each instance the epithelial membrane extended into the anterior chamber from the fistula.

Each case was treated by Maumenee's technique, consisting of removal of the membrane by curettage and cauterization of the curetted area with 70% ethyl alcohol. The first case, with minimal involvement, obtained an excellent result. The second case is apparently a functional failure. The third case developed sympathetic ophthalmia necessitating enucleation. It is uncertain whether the sympathetic ophthalmia resulted from the initial cataract surgery with iris incarceration or from the operation for the removal of the epithelial downgrowth.

Conclusions

Surgical removal of epithelial downgrowth in the anterior chamber is probably indicated in early cases without extensive involvement.

This procedure accomplishes closure of a postoperative fistulous tract.

Accurate histological diagnosis by biopsy can be obtained.

The operation should be regarded as a major ocular procedure and is not without hazard.

Epithelization of the anterior chamber is such a serious complication that heroic measures such as this may be justified.

Dr. George Brown, of Greeley, Colo., referred Case 1; Dr. Ralph Danielson, of Denver, referred Case 2, and Dr. Royce Tebbet, of Casper, Wyo., referred Case 3.

Metropolitan Bldg. (2).

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Cataract Extraction and Aqueous Outflow

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Glaucoma after cataract extraction occurs more frequently following operative and postoperative complications.[1,2] Thus, the presence of retained lens material, a flat anterior chamber, iridocyclitis, or vitreous loss results in an increased incidence of peripheral anterior synechiae and glaucoma. Rupture of the lens capsule appears to be the complication which leads to glaucoma most frequently. It was the purpose of the present study to attempt to evaluate the effects of cataract extraction and its complications on the outflow channels. It was thought that pre- and postoperative tonography and gonioscopy might offer an approach whereby more subtle changes in aqueous outflow could be detected even in the absence of overt glaucoma.

Methods

A group of 111 patients were subjected to repeated tonography and gonioscopy before and after cataract surgery. These persons had a total of 139 cataract extractions performed by house officers of the Washington University Clinic Service between Sept. 8, 1955, and Jan. 25, 1957. During this same interval approximately 500 cataract operations were carried out on this service. Only those patients were selected who met the criteria described below and who could be followed closely in the postoperative period. This resulted in a series of carefully studied eyes with a higher percentage of complications than in the total cataract series. Fifteen eyes of 11 patients with preexisting unoperated-on chronic simple glaucoma and cataracts were included in the present series.

Tonography was performed one or more days prior to surgery with an electronic tonometer connected to a Leeds and Northrup recorder. Calculations for the coefficient of facility of outflow were made with use of the 1955 Friedenwald tables.[3] The chamber angles were studied and charted with use of the Zeiss slit lamp with gonioscopic attachment. All patients with synechiae before surgery or with any evidence of inflammatory or neoplastic disease were excluded from the series. Operation was performed with either a limbal incision and a limbal-based flap or with a section anteriorly in clear cornea. Round pupil intracapsular extractions were attempted in all cases. Ruptured capsules were recorded as such even if all visible lens was removed. In the event of vitreous loss an iridectomy above and radial incisions in the sphincter below were performed routinely. Four to six weeks postoperatively tonography and gonioscopy were repeated. Uncomplicated cases were then followed at two-month intervals. Suspicious cases had more frequent observations. Follow-up periods varied from 3 to 18 months.

Postoperatively a significant number of patients demonstrated an apparently decreased intraocular tension as measured with a tonometer. Since decreased scleral rigidity might also manifest itself similarly, rigidity measurements were made on 20 eyes at different intervals. Comparisons were made

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of intraocular pressure measurements with use of the tonometer with various weights and the Goldmann aplanation apparatus. Plotting these readings revealed the scleral rigidity to be within the normal limits seen in a general clinic population. Six of the patients who had monocular cataract extractions could have comparisons made of the operated-on and unoperated-on eyes. Four of these showed slightly increased rigidity in the operated-on eye compared with the contralateral eye, and in two eyes rigidity was decreased to a minimal degree. On the basis of these determinations, alterations in scleral rigidity were disregarded throughout the remainder of the series.

The complications recorded and tabulated were rupture of the lens capsule, hyphema, any shallowing or a flat anterior chamber, vitreous loss, iris prolapse, and endophthalmitis. All subjects were examined postoperatively for synechiae, decreased coefficient of outflow facility, and other evidence of glaucoma. Alterations in these observations which were arbitrarily set as moderate changes for purposes of this series were synechiae involving more than one-fourth of the chamber angle or a decrease of outflow facility coefficient of more than 25%. Marked alterations were defined as more than one-half the angle occluded by synechiae or the outflow facility decreased by 50% or more. Glaucoma was diagnosed by persistent elevations of intraocular pressure over 24 mm. Hg associated with flattening of the tonographic tracing.

Results

The eyes of almost all postoperative cataract patients demonstrated a state of hypotony and an associated decreased facility of outflow. The recovery of both secretion and outflow facility occurred following a variable period of from less than six weeks to over a year. Two patients have persisted in their hypotonic state for longer than 15 months. Eyes with preexisting chronic simple glaucoma did not seem to differ from the nonglaucomatous eyes in the incidence or duration of hypotony.

In the entire series of 139 eyes there were 40 (29%) which had synechiae involving more than one-fourth of the angle, and 51 eyes (37%) demonstrated more than a 25% decrease in facility of outflow. Extensive synechiae (more than half the angle) occurred in 12 eyes (9%), and outflow facility was decreased 50% or more in 19 eyes (14%). Of the 124 eyes with no evidence of glaucoma preoperatively, 9 (7%) developed frank glaucoma (Table). The site of the section did not appear to alter the incidence or extent of synechiae nor the severity of the facility changes.

In the group of 78 eyes which had cataract extraction without detectable complication there was a very much lower incidence of synechiae and decreased outflow facility. Of this group only 14 eyes (18%) had more than one-fourth the angle occluded by synechiae and 22 eyes (28%) demonstrated moderately impaired outflow facility. More than half the angle was occluded by syn-

Incidence of Disordered Outflow Following Cataract Surgery

Complication	Eyes, No.	Moderate Change *				Marked Change †				Glaucoma	
		Synechiae		Decreased Outflow Facility		Synechiae		Decreased Outflow Facility			
		No.	%	No.	%	No.	%	No.	%	No.	%
Total series	139	40	29	51	37	12	9	19	14	9/124	7
None	78	14	18	22	28	2	3	6	8	1/66	1.5 ‡
Ruptured capsule	23	10	43	12	52	7	30	8	35	6/20	30
Hyphema	21	7	33	10	48	2	10	3	14	1/21	5
Shallow anterior chamber	15	7	46	7	46	4	27	4	27	2/14	14
Vitreous loss	11	6	54	5	45	3	27	3	27	2/11	18
Iris prolapse	3	3		2		0		0		0	
Infection	1	1		1		1		1		1	
Previous glaucoma	15	2	13	5	33	1	7	1	7	--	

* Synechiae involving more than one-fourth of the angle or greater than 25% decrease in outflow facility (includes marked changes).
† Synechiae involving more than one-half of the angle or 50% or greater decrease in outflow facility.
‡ Subsequently developed chronic simple glaucoma in unoperated-on, contralateral, eye.

echiae in only two eyes (3%) of this group, and markedly impaired outflow facility was apparent in six eyes (8%). Of the 66 eyes in this uncomplicated group, which did not have evidence of preexisting glaucoma, only one eye developed glaucoma postoperatively. Interestingly enough, only approximately one-third of the angle was occluded by synechiae in this eye. Although a single tonographic tracing before surgery was within normal limits in both eyes, the patient subsequently developed chronic simple glaucoma in his unoperated-on, contralateral, eye. Thus, the operated-on eye might well have been classified as having glaucoma preoperatively had suitable provocative tests been carried out before surgery.

There were 23 eyes available for study in which the capsule ruptured during the attempted intracapsular extraction. Six of these eyes (30%) developed secondary glaucoma. In two eyes the glaucoma resulted from incomplete extraction as the sole complication. In both instances, however, considerable amounts of lens material remained in the eye at the close of the procedure. The other four eyes in this group which developed glaucoma had additional complications, such as vitreous loss, flat anterior chamber, or hyphema. The incidence of moderate degrees of synechiae and reduction of outflow facility was considerably higher in the group with ruptured capsule than in those eyes with uncomplicated extractions. There were 10 eyes (43%) with synechiae of more than one quadrant and 12 eyes (52%) with moderately decreased coefficients of outflow facility following extracapsular extraction. Markedly impaired outflow facility and extensive synechiae occurred much more commonly (30%-35%) in this group than following any other complication.

Hyphema of variable magnitude occurred in 21 eyes. A moderate number of synechiae were seen in 7 (33%) of these, and decreased facility of outflow of more than 25%, in 10 (48%). Marked changes in facility occurred in three eyes (14%) and extensive synechiae in two eyes (10%). In only one eye did hyphema result in glaucoma. This was in a patient who had oculogyric crises postoperatively and suffered a massive hyphema. The anterior chamber remained filled with blood for a considerable period of time, resulting in an elevated pressure in the early postoperative period. Following resorption of the hyphema, extensive synechiae, decreased facility of outflow, and glaucoma persisted in this eye.

Shallow or flat anterior chambers occurred in 15 eyes which could be studied both pre- and postoperatively. In all of these the anterior chambers were not permitted to remain flat for more than four or five days before air injections were carried out. In spite of this precaution, the incidence of peripheral anterior synechiae and reduction of facility of outflow were considerably higher in this group than in the uncomplicated series. This was true when the moderate changes in outflow channels (in 46%) as well as the marked changes (in 27%) were considered. Two of the fourteen eyes in this group without preexisting glaucoma developed glaucoma postoperatively.

The effects of loss of vitreous could be studied in 11 eyes. Synechiae involving more than one-fourth of the angle and moderate changes in outflow facility occurred in approximately half of these eyes. Marked changes in both of these parameters appeared in 3 (27%) of the 11 eyes in this group. Two eyes developed secondary glaucoma.

Of interest were the three eyes with iris prolapse. None of these developed extensive synechiae, marked facility changes, or secondary glaucoma, even though one had an associated complication of vitreous loss at the time of prolapse. However, all three eyes had synechiae and two presented moderately decreased facility of outflow.

One patient developed an endophthalmitis on the third postoperative day. Although the infection was brought under control, the

eye developed extensive synechiae, markedly impaired outflow facility, and secondary glaucoma.

The 15 eyes available for study which had a preoperative diagnosis of chronic simple glaucoma had no greater occurrence of synechiae or alterations of outflow facility than did the rest of the series. In the glaucoma group, as in the normal group, synechiae and decreased outflow facility were commoner in eyes with operative or postoperative complications.

Comment

In addition to providing valuable data as to the effects on the outflow channels of cataract surgery and its complications, preoperative tonography aided in the detection of unrecognized glaucoma. Unsuspected chronic simple glaucoma was discovered in 4 patients (3.9%) of the 104 not previously so diagnosed in this series. A decreased facility of outflow on the routine tonogram suggested the diagnosis, which was then substantiated by water provocative tests or loss of visual field.

One of the commonest findings in this series was the period of hyposecretion that followed cataract surgery. This confirmed Hilding's observations of hypotony persisting for variable periods after operation.[4] In the present series, hyposecretion lasted from less than 6 weeks to over 15 months and was usually associated with a decreased facility of outflow. Realization that this phenomenon occurs is important. On the one hand, the reduced facility of outflow is not to be mistaken for a glaucomatous state. The intraocular pressure in these eyes is extremely low, and P_o/C values are well under 100.[5] Furthermore, the altered facility recovers with the return of secretion and is probably a compensatory phenomenon. On the other hand, when one follows patients with chronic simple glaucoma during their postoperative interval, the hyposecretion of aqueous can normalize intraocular pressure and lead to a false sense of security. It is necessary to observe such patients at frequent intervals in order to recognize the resumption of normal secretion and to reevaluate the status of glaucoma control. It is also apparent that previously well regulated glaucoma may be thrown out of control by a reduction in outflow capacity of insufficient magnitude to affect a normal eye. In the present series of 15 eyes with chronic simple glaucoma, 4 eyes were uncontrolled with their preoperative medication following cataract surgery. These required more intensive medical therapy or surgery.

It has been observed previously that the majority of eyes demonstrate peripheral anterior synechiae following cataract surgery even in the absence of complications.[2] When synechiae of all degrees were tabulated in the present series, the incidence was 74% (103 eyes). It is a fortunate circumstance that this scarring does not always result in much damage to outflow channels. However, it is of considerable interest that following uncomplicated cataract extractions many eye have more extensive synechiae and a considerable decrease in outflow facility as well, but of insufficient degree to induce secondary glaucoma. It is only in those instances where the synechiae and decreased outflow facility are marked that glaucoma becomes a frequent occurrence. Even in some eyes with extensive impairment of outflow channels, intraocular pressure may not be elevated. In these eyes glaucoma is apparently avoided by persistent hyposecretion. It should be emphasized, however, that pressure rises may be anticipated with return of secretion of aqueous. In these instances of potential glaucoma the damage must be evaluated by gonioscopy and tonography and the patients followed closely.

Secondary glaucoma has been reported to be three times as prevalent in extracapsular extractions.[1] In the present series, the incidence of moderate decreases in outflow facility and synechiae of more than one quadrant was doubled following rupture of the lens capsule as compared with uncomplicated extraction. More extensive damage

to outflow channels occurred very frequently in the group with lens remains, and 30% of all eyes with ruptured capsules developed secondary glaucoma.

Hyphemas following cataract surgery have been considered to have negligible effect on the ultimate visual result.[6,7] In this study anterior chamber bleeding did increase the incidence of lesser degrees of synechiae and of decreased outflow facility. However, more extensive changes were less frequent and rarely of sufficient degree to result in a significant incidence of glaucoma.

Kronfeld and Grossman[2] have reported that extensive synechiae were present in 32 eyes with aphakic glaucoma which had flat chambers for more than five days following cataract surgery. It is of interest that 23 of the eyes in their glaucoma series had ruptured capsules as well as flat anterior chambers. In the series reported in the present paper both shallow and flat chambers were re-formed by wound suturing and air injection if they persisted for five days. In spite of this short duration, the incidence of synechiae, decreased facility, and glaucoma was greatly increased over that in the uncomplicated group of cataract extractions.

Vitreous loss has been described as predisposing to the development of glaucoma. No definite conclusions can be drawn from the few eyes available in this series where this complication occurred by itself. However, impaired outflow facility and synechiae were a frequent consequence of vitreous loss.

Of the complications considered, the extreme of any of them produced the greatest damage. In other words, the outflow disorder tended to correlate with the amount of lens material left in the eye, the severity of the hyphema, etc. Combinations of several complications tended to further increase the incidence and severity of synechiae and decreased outflow facility. In general, the reduction of outflow facility could be correlated with the extent of the peripheral anterior synechiae, but this generalization did not apply to each individual eye. Both of these alterations occurred more frequently than was anticipated from the incidence of secondary glaucoma.

Summary and Conclusions

A variable degree of damage to the aqueous outflow mechanism is a common occurrence following cataract extraction. Even in apparently uncomplicated cataract surgery, peripheral anterior synechiae and reduction of facility of outflow occur but are rarely of sufficient degree to produce glaucoma.

Complications at the time of surgery and in the immediate postoperative period increase the incidence and severity of synechiae and impairment of outflow facility. When extensive enough, these result in secondary glaucoma. Rupture of the lens capsule at the time of surgery is the most prominent offender, resulting in a 30% incidence of glaucoma and accounting for two-thirds of the secondary glaucoma in this series.

Cataract extraction is followed by a state of hyposecretion of aqueous humor lasting from less than 6 weeks to more than 15 months. The decreased rate of secretion lowers intraocular pressure and makes it difficult postoperatively to appreciate the obstruction to aqueous outflow by tonometric measurements alone.

Eyes with preexisting chronic simple glaucoma and normal eyes behave similarly in respect to alterations in gonioscopic and tonographic findings after cataract extraction.

Sections in clear cornea or at the limbus under limbal-based flaps did not result in measurable differences in tonographic or gonioscopic alterations.

In cataract surgery, routine tonography and gonioscopy pre- and postoperatively offer opportunities for the detection of unrecognized glaucoma as well as an evaluation of the damage to outflow channels induced by the operation.

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Tonographic and Gonioscopic Studies Before and After Cataract Extraction

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Tonographic studies of eyes with aphakic glaucoma have shown impairment of aqueous outflow. There has been, however, little knowledge as to what outflow facility prevailed in such eyes before the lens was extracted and as to the effect of cataract extraction on outflow facility in eyes not developing clinically recognized aphakic glaucoma. The purpose of this paper is to present a comparison of tonographic and gonioscopic studies made prior to and at intervals following cataract extraction in a series of 142 eyes (in 124 patients, 18 of whom had both lenses removed).

Method

The diagnosis of cataract was made in the Eye Clinic at the Massachusetts Eye and Ear Infirmary. No topical medication had ben instilled during the 48 hours before preoperative tonography. All tonographic and gonioscopic studies were done by one of us (P. F. L.). Tonography, practiced according to the method of Grant, was done at (approximately) 3, 6, 12, and 24 weeks after lens extraction, and gonioscopy was done at least once in the postoperative period (and always at the time elevated tension was first recognized).

All measurements of intraocular pressure in this study were made with the electronic tonometer (V. Mueller Company) and expressed according to the 1948 Calibration of the Committee on Standardization of Tonometers. All tonographic tracings were made on a Sanborn recorder. Facility of aqueous outflow (C) was calculated from the 1954 Calibration of the Committee on Standardization of Tonometers; in calculating aqueous flow (F) the pressure in recipient veins was considered to be 10 mm. Hg.

Most of the cataract extractions were done by senior residents, as follows: After limbal peritomy, three tract sutures were placed; a knife incision was made, and the tracts were rethreaded with 000000 mild chromic absorbable surgical (gut) sutures before (or occasionally after) the wound was enlarged with scissors. The lens was either grasped with Verhoeff capsule forceps and slid or with an erysiphake and tumbled, after a full iridectomy was made (occasionally a peripheral iridectomy).

I. Tonographic Findings

A. Tonographic Findings in Unoperated-On Contralateral Eyes.—In 105 unoperated-on contralateral eyes, the average value for facility of outflow and rate of flow remained unchanged after the fellow eye had received a cataract extraction. (One contralateral eye was ignored because of its long-standing absolute, hemorrhagic glaucoma.)

B. Tonographic Findings in the Entire Group of Eyes Whose Lenses Were Extracted.—In the entire group of 142 cataractous eyes from which the lenses were extracted, tonography before and after operation gave the values shown in Table 1.

1. Eyes Demonstrating Cataracts but No Glaucoma or Other Pathology: No pathol-

From the Massachusetts Eye and Ear Infirmary.

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TABLE 1.—*Results of Tonography in 142 Cataractous Eyes*

	Av. Facility of Outflow (C), Cu. Mm./Min./Mm. Hg.	Av. Rate of Aqueous Flow (F), Cu. Mm./Min.
Before operation	0.23	1.57
After operation, wk.		
3	0.18	1.12
6	0.19	1.08
12	0.20	0.97
24	0.18	1.04

ogy other than cataract was found in 115 eyes; after cataract extraction, three patterns of tonographic behavior were observed in these 115 eyes: Outflow facility (C) decreased in 61, remained the same in 36, and increased in 18.

(*a*) Early decreased outflow facility. Sixty-one eyes showed before cataract extraction average outflow facility of 0.23±0.01. Postoperatively, C averaged 0.13±0.01. Although all 61 showed some decrease in C, 7 of these eyes fell to the glaucoma borderline area, and 28 decreased so much as to enter the frankly glaucomatous range at the time of first postoperative tonography (Fig. 1). A comparison of the behavior of these 28 eyes with that of their unoperated-on fellow eyes is of interest (Table 2; Graph 1).

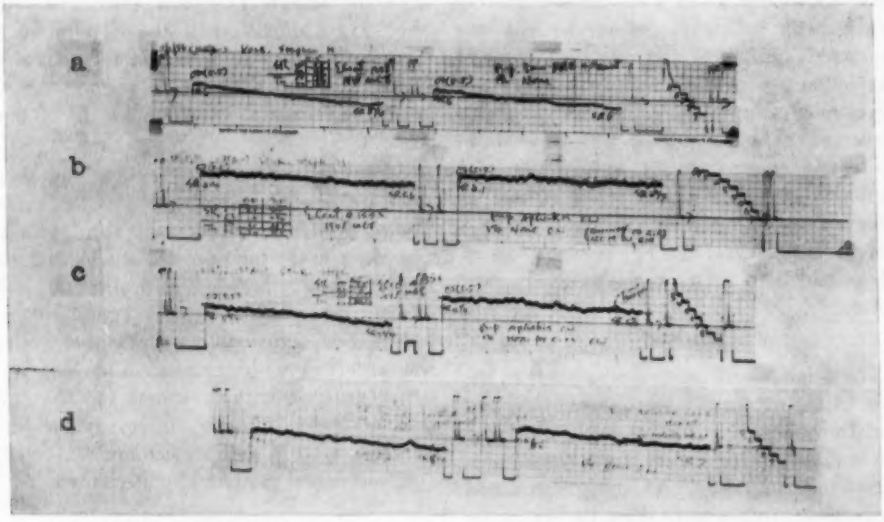
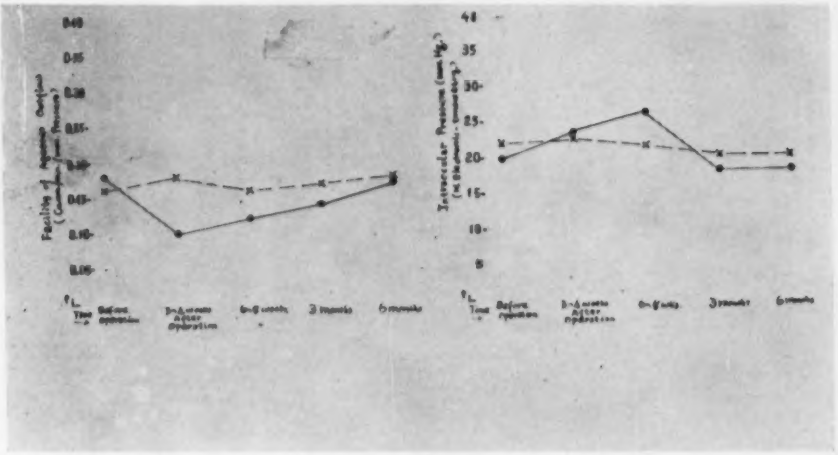


Fig. 1.—Tonographic tracings from a patient who received bilateral intracapsular cataract extraction, showing (*a*) normal preoperative tension and outflow facility O. U.; (*b*) elevated tension (O. D. 30, O. S. 32) and impaired C, O. U., 5 weeks after lens extractions; (*c*) normal tension (O. D. 18, O. S. 24); C normal in O. D., but still impaired in O. S., 8 weeks after lens extractions; (*d*) normal tension (O. D. 15, O. S. 23) and normal C in both eyes, 16 weeks after lens extractions.

(*b*) Unchanged outflow facility. Twenty-nine eyes showed no significant change in

TABLE 2.—*Early Decreased Outflow Facility*

	28 Eyes Whose C Fell Postoperatively into the Glaucoma Range			28 Contralateral (Unoperated-On) Eyes	
	Av. IOP	Av. C	Av. F	Av. C	Av. F
Before operation	19.9	0.18	1.43	0.16	1.67
After operation, wk.					
3	23.3	0.10	1.02	0.18	1.73
6	26.0	0.12	1.12	0.16	1.50
12	18.0	0.14	1.00	0.17	1.48
24	18.3	0.17	1.21	0.18	1.78



Graph 1.—Left, curve shows average C of 28 eyes from which the lens was extracted and of the unoperated-on fellow eyes; C was measured before operation and at (approximately) 3, 6, 12, and 24 weeks after operation. C showed an early decline into the glaucoma range in the operated-on eye, followed by gradual return to normal levels. Right, curve shows ocular tension in the same 28 operated-on eyes and their unoperated-on fellows. An early rise in tension in the operated-on eyes was followd by a return to normal tension without treatment. Black dot, operated-on eyes; X, unoperated-on eyes.

outflow facility after their lenses had been extracted:

	Av. C
Before operation	0.20
After operation, wk.	
3	0.22
6	0.20
12	0.19
24	0.20

(*c*) Late decrease in outflow facility. Seven eyes which had shown no change in C by earlier tonography demonstrated a late decrease from an average value of 0.24 (at 3 weeks after lens extraction) to an average value of 0.13 (at 12 to 20 weeks after operation) but without a corresponding rise in average intraocular pressure (Graph 2):

	Av. C
Before operation	0.22
After operation, wk.	
3	0.24
8	0.21
12-20	0.13

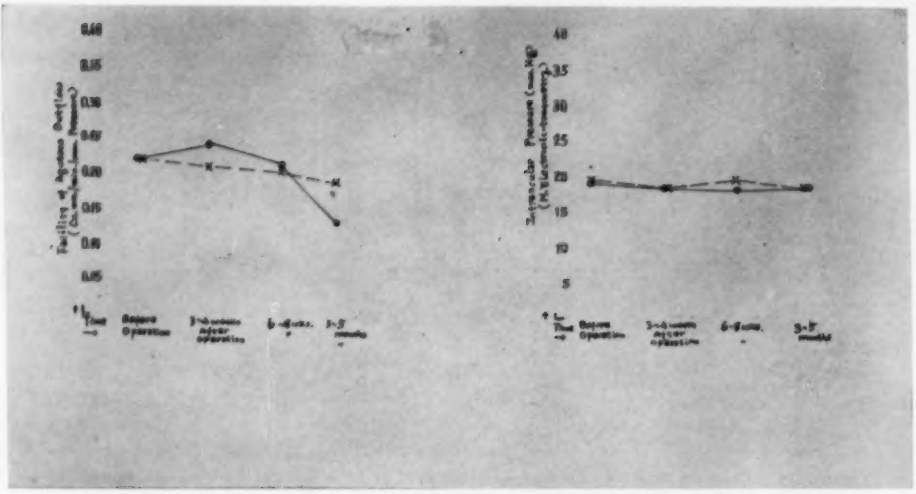
(*d*) Increased outflow facility. Increased outflow facility was observed after cataract extraction in 18 eyes (Table 3).

Although the increase in C value was generally transient, two eyes continued to show increased outflow facility three months after lens extraction.

(*e*) Outflow facility not measurable. There were 13 eyes which, after lens extraction, were so soft that tonography was impossible for a period of six weeks or, in some cases, longer (Fig. 2).

2. Eyes Known to Have Glaucoma Prior to Lens Extraction: In seven glaucomatous eyes the average facility of outflow was 0.14 before cataract extraction and was 0.11 at 3 to 4 weeks after operation, 0.13 at 5 to 6 weeks after operation, 0.11 at 12 weeks after operation, 0.14 at 16 weeks after operation, and 0.16 at 24 weeks after operation (Graph 3). Four of these eyes had glaucoma capsulare; two had subacute angle-closure glaucoma, and one had open-angle glaucoma.

C. Tonographic Findings in Eyes with Postoperative Complications.—1. Increased Intraocular Pressure: Thirty-five eyes in 30 patients were found to have elevated intraocular pressure at some time during the six-month postoperative period. Seven of these eyes (six patients) had glaucoma preoperatively. Of the remaining 28 eyes, facility of outflow and ocular tension returned spontaneously to the normal range in 25; in 1, tension and C are now normal on



Graph 2.—Left, curve shows late decline in C of seven eyes from which cataract was extracted, but there is essentially no change in the unoperated-on fellow eyes. Right, curve shows, in the same seven eyes, that a rise in ocular tension did not accompany the late fall in C. Black dot, operated-on eyes; X, unoperated-on eyes.

no treatment, although antiglaucoma therapy was required for a time, and in 2 pressure has fallen to normal but C remains low.

2. Hyphema: Of nine eyes developing hyphema after lens extraction, seven showed early absorption of the blood without damage to the eye and with return of outflow facility to normal. The hyphema was accompanied, in another eye, by glaucoma which later subsided without treatment, with a return to normal outflow facility. The ninth eye with hyphema developed hemorrhagic glaucoma.

3. Flat Anterior Chamber with Separation of Choroid: Lens extraction was followed in 10 eyes by flat anterior chamber with choroidal separation. Two of these ten eyes developed secondary glaucoma as a result of complete peripheral anterior synechias (one of the two had been treated for open-angle glaucoma preoperatively). The average C, F, and tension before and after lens extraction in the remaining eight eyes which developed flat anterior chamber, (but no synechias) offered no explanation of the mechanism of choroidal separation.

4. Rupture of Lens Capsule: Eleven eyes received unplanned extracapsular cataract extractions; six of these were essentially intracapsular procedures, inasmuch as the lens capsule was broken while the lens was being delivered, and the operator observed that no material was left in the anterior chamber. Outflow facility in this group behaved thus:

	Av. C
Before operation	0.22
After operation, wk.	
3	0.16
6	0.17
12	0.23
24	0.19

TABLE 3.—*Increased Outflow Facility*

	18 Cataractous Eyes		18 Unoperated-On Fellow Eyes
	Av. C	Ocular Tension	Av. C
Before operation	0.23	17.7	0.23
After operation, wk.			
3	0.32	14.3	0.22
6	0.27	15.0	0.22
12	0.25	16.5	0.20

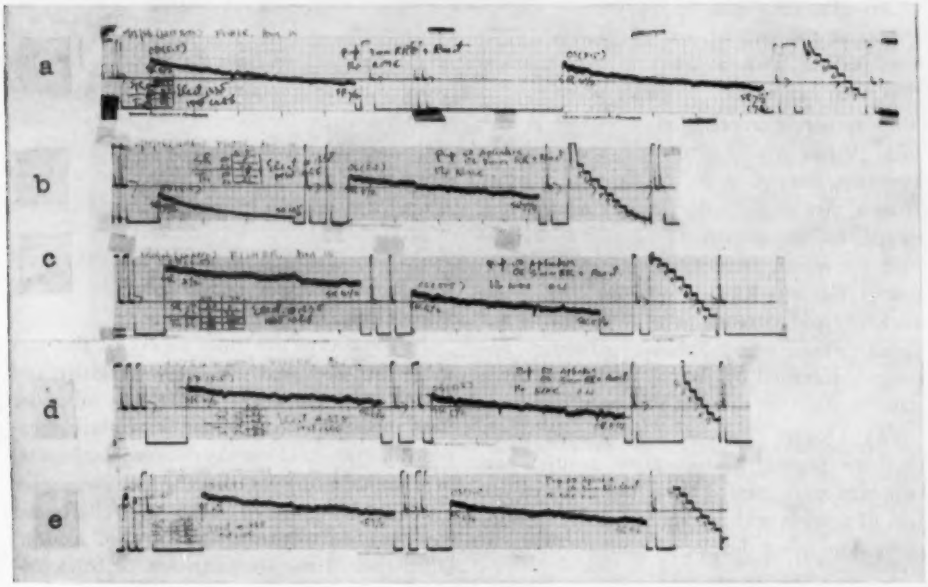
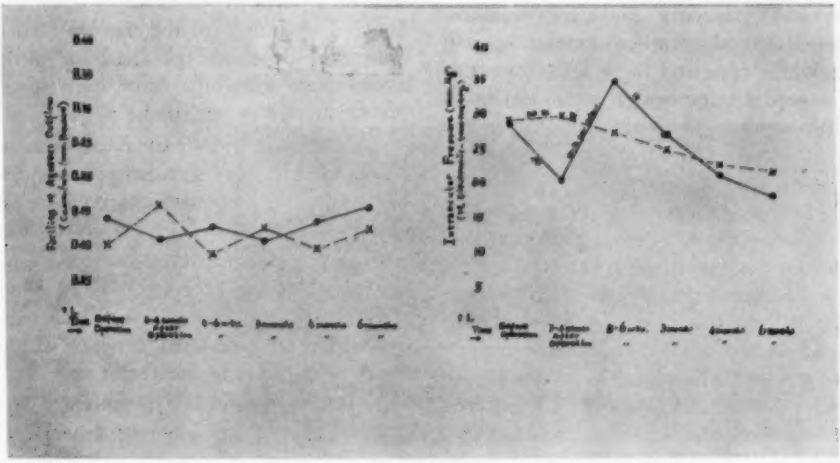


Fig. 2.—Tonographic curves of both eyes made before and at 4, 7, 11, and 19 weeks after a cataract was extracted from O. D. On the right, above, are seen curves from the unoperated-on eye; all showed normal outflow facility. On the left-hand side, above, curves from the operated-on eye show (*a*) normal C in O. D. before operation; (*b*) 1 month after operation, O. D. so hypotonic that C was incalculable; (*c*) 7 weeks after operation, C had fallen in O. D. to the glaucoma range and tension had risen to 31 mm. Hg; (*d*) and (*e*) C and tension returning to normal at 11 and 19 weeks, respectively, after the lens was extracted from O. D.

Graph 3.—Left, curve shows outflow facility of seven glaucomatous eyes before and at intervals after they had received cataract extractions; curve also shows C for the glaucomatous fellow eyes which were not operated on. Right, curve shows, after lens extraction, an early fall and later rise in the ocular tension of eyes which, before cataract extraction, were known to have glaucoma. The tension curve for the operated-on eyes shows a still later fall as a result of treatment. Black dot, operated-on eyes; X, unoperated-on eyes.



No glaucoma had been observed in this group at the end of six months except in one patient with a vitreous complication, described below.

5. Other Complications: (*a*) Vitreous loss. Vitreous was actually lost during the operation in two eyes; neither eye showed tension rise or reduction in C during a six-month follow-up period. In one additional case (in which there was preoperative glaucoma) the lens capsule was ruptured inadvertently and vitreous presented; during the convalescent period aphakic glaucoma appeared, followed by retinal separation three months after the cataract had been removed.

(*b*) Uveitis. Two eyes developed more than the normal postoperative uveitis; one case was accompanied by a transitory elevation of tension with impaired facility of outflow. The other developed phthisis bulbi.

II. Gonioscopy and Associated Outflow Findings

One hundred twenty of the one hundred forty-two cataract eyes received at least one postoperative gonioscopic examination.

A. Preoperative Findings.—Preoperative gonioscopic examination revealed two eyes to have narrow angles; they had, in fact, subacute angle-closure glaucoma. Eleven cases revealed so-called exfoliation of the lens capsule, some with capsular material visible on the filtration meshwork; four of these had glaucoma capsulare, and two others developed, after lens extraction, glaucoma which appeared to be transitory. One eye showed a large benign melanoma in the angle; another showed an old trabecular synechia, presumably the result of inflammation, and another showed evidence of old posterior synechias. One patient with diabetic retinopathy showed early rubeosis of the iris; after lens extraction this eye developed hemorrhagic glaucoma. Fifty-four eyes showed enough pigment in the form of dust or clumps in the lower angle to be noteworthy, and twelve eyes showed a posterior meshwork sufficiently discolored to be remarked.

B. Postoperative Findings.—1. Inner Scar: The usual surgical scar remaining after cataract extraction was seen by gonioscopy to have its inner aspect located anterior to Schwalbe's line at 12 o'clock by about the same distance that separates Schwalbe's line from scleral spur. The 3 o'clock and 9 o'clock extremities of the inner scar (where enlargement by scissors had taken place) were seen usually to lie nearer Schwalbe's line, however, than did the inner scar at 12 o'clock.

In some eyes the inner aspect of the surgical scar appeared unusually irregular and ragged and the edges sometimes failed of neat apposition because one lip slightly overrode the other. Descemet's membrane was, in a number of eyes, detached at the region (usually 10 and 2 o'clock) where scissors were applied to extend the surgical section. Distortion of the scar attributable to excessively tight sutures was noted in two eyes. Partial invasion of the filtration meshwork by the surgical scar was noted in eight eyes.

In no case could the character or location of the scar, in itself, be indicted as producing any significant change in C, F, or intraocular pressure, except when there was manifest incarceration of iris pillar in the wound (see below).

2. Synechias: Peripheral anterior synechias extending so far circumferentially as to occlude the angle completely were seen in 2 of 10 eyes in which flat anterior chamber and separation of the choroid occurred; both eyes developed glaucoma as a result (open-angle glaucoma had been observed preoperatively in one of the two).

Adhesion of iridectomy pillars was seen in 68 of 120 eyes gonioscoped postoperatively, but this was not observed to affect outflow facility.

Incarceration of iris pillar in the wound was observed in 12 eyes. All 12 showed increased facility of outflow, which varied, apparently, in individual patients with the size and character of incarceration.

In 18 eyes incomplete iridectomy left iris root which became adherent anteriorly to

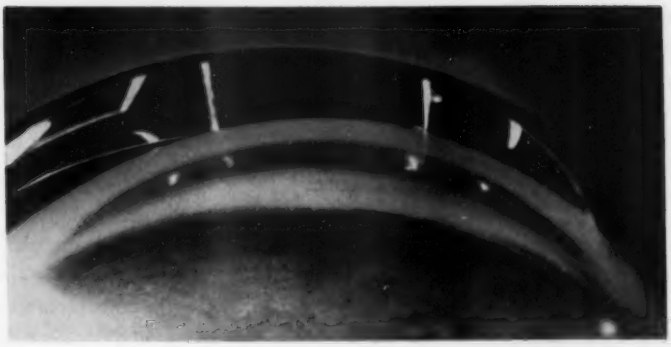


Fig. 3A.—Goniophotograph showing rather dense pigment deposition in the inferior angle six weeks after lens extraction; C was depressed, and ocular tension was elevated.

ciliary body band, scleral spur, or trabeculum. In six eyes small synechias were observed to be attached to ciliary body band, sometimes as far forward as scleral spur; in eight eyes there were small synechias attached to the trabecular meshwork above. In 12 eyes ciliary processes appeared to herniate forward slightly either for no apparent reason or because they were drawn forward by vitreous which was adherent to the scar. No significant difference in C, F, or ocular tension was observed in any of these eyes.

3. Deposition of Pigment or Debris on the Filtration Meshwork: Twenty-six eyes showed increased pigment deposition in the inferior angle after lens extraction; the pigment was observed in varying amounts in different eyes, and there appeared to be no relationship between its density and the occurrence of synechias or other event. Only when there was unusually wide-spread and dense pigment deposition in the angle and on the meshwork did outflow facility appear to decline and ocular tension rise. In one case which developed poor outflow and increased pressure there occurred, 16 weeks after operation, spontaneous improvement in C and lowering of tension; the improvement seemed related to partial disappearance of the pigment dust in the entire angle (Figs. 3A and 3B). This patient was not diabetic.

4. Vitreous: Vitreous was seen to be attached to the inner scar (24 eyes), to be incarcerated in the wound (5 eyes), or to be herniated into the anterior chamber (9 eyes). In none of these eyes was any significant change in outflow facility or rate of flow observed.

5. Miscellaneous Gonioscopic Findings: In seven eyes cotton fibrils were observed in the anterior chamber after operation. No untoward effect on the eye was noted,[8] whether the fibril was in contact with corneal endothelium, inner scar, iris, or hyaloid membrane.

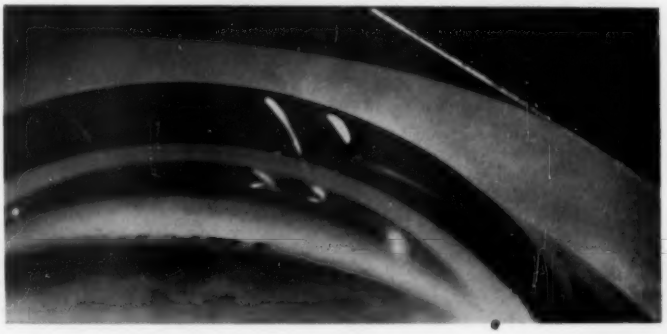


Fig. 3B.—Goniophotograph of the same angle as in Figure 3A 20 weeks after lens extraction, showing disappearance of some pigment; tension and C were now normal (no treatment had been used).

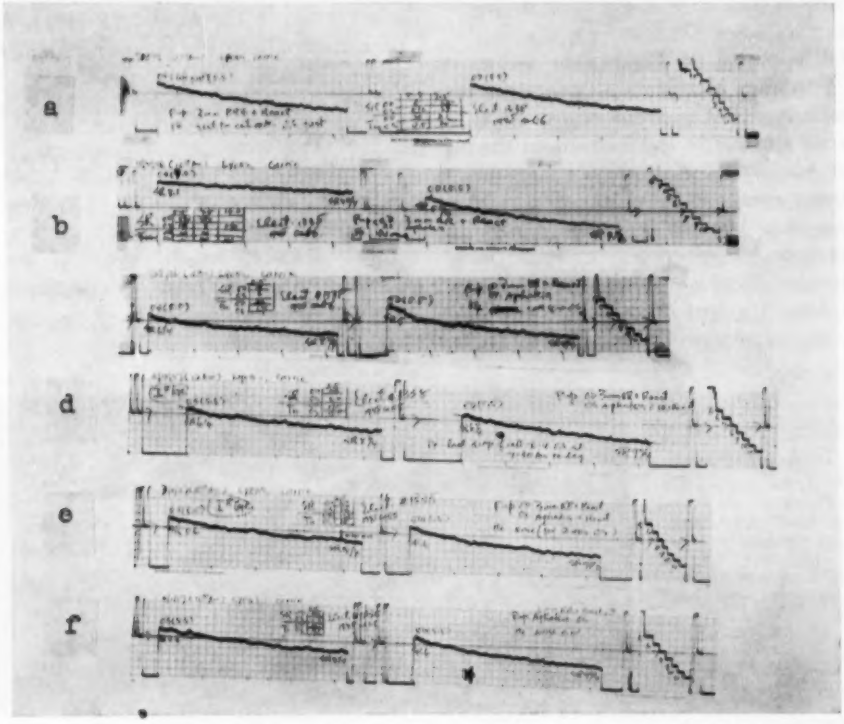
Comment

Grant [1] stated that the differences between normal unoperated-on eyes and uncomplicated aphakic eyes are slight and possibly insignificant, either in average rate of aqueous formation or in average value of facility of aqueous outflow; he did not compare the average values of aqueous formation and the facility of outflow in the same eye before and after lens extraction. Scheie and his associates [2] reported that the facility of aqueous outflow is diminished in eyes with glaucoma after cataract extraction; however, they did not mention the duration of postoperative evaluation or at what time in the postoperative period the facility of outflow was diminished. Von Fieandt [4] reported gonioscopic findings after lens extraction.

According to our findings, the facility of aqueous outflow of most eyes is diminished after cataract extraction; this diminution is usually evident 3 weeks after operation and usually is present only temporarily, returning to normal usually at about 12 weeks, but as late as 24 weeks in some eyes. On the other hand, there is a smaller number of eyes whose facility of outflow remains unchanged or is increased after cataract extraction; when increased facility was demonstrated, incarceration of the iris was generally visible.

An appearance suggestive of diffuse edema of the inner aspect of the wound

Fig. 4.—Tonographic curves from both eyes of a patient who received a lens extraction from O. S. On the right-hand side, above, are shown curves revealing C to be normal both preoperatively and postoperatively in the unoperated-on eye. On the left-hand side, above, tonographic curves from the operated eye show (*a*) normal C and tension before lens extraction; (*b*) tension 40 mm. Hg and glaucomatous C 5 weeks after lens extraction; (*c*) and (*d*) tension and C normal on pilocarpine therapy at 9 and 20 weeks after O. S. had its lens removed; (*e*) and (*f*) normal tension and C after pilocarpine was discontinued (curves made at 23 and 27 weeks, respectively, after lens extraction O. S.).



lips, especially on the scleral side, was noted by gonioscopy in a number of eyes; this phenomenon was evident only in eyes with increased intraocular pressure and persisted sometimes for several weeks. When tension and C had returned to normal, the swelling had generally disappeared.

We have been interested in the possible mechanism of flat anterior chamber with choroidal detachment after cataract extraction. Unfortunately, there are no definite predisposing factors which we can recognize in the explanation of its mechanism. Although seven eyes which showed preoperatively a relatively low rate of aqueous formation developed flat anterior chambers after lens extraction, three others showed no such low formation rate. Fluorescein tests for wound leak were negative in 9 of the 10 eyes with flat chambers.

One case of glaucoma resulting from closure of approximately 90% of the angle by synechias (following flat anterior chamber) is of interest: In this case, some of the peripheral anterior synechias were observed to have disappeared following treatment with 2% pilocarpine, leaving the full width of posterior meshwork exposed for half its circumference; both intraocular pressure and facility of outflow returned to normal and remained there after treatment was discontinued (Fig. 4). It would seem, therefore, that early gonioscopic examination should be done routinely after a flat chamber has reformed; early institution of miotic therapy might be justified whether or not there is early tension elevation, if peripheral anterior synechias are present.

Another case which deserves special comment is one of secondary glaucoma associated with postoperative uveitis. In this case, intraocular pressure rose to above 35 mm. Hg and tonography showed definite impairment of outflow facility. No synechias were visible. Both intraocular pressure and facility of outflow returned to the normal range gradually and spontaneously during the first four months after operation, without antiglaucoma therapy. Six months later, the facility of outflow and intraocular pressure were still within normal limits.

Two eyes had not been recognized to have glaucoma until increased ocular tension was measured at the time of preoperative tonography; by gonioscopic examination the diagnosis of subacute angle-closure glaucoma was made. After lens extraction and full iridectomy the angles became wide, and, without medication, tension and C thereafter remained normal in these eyes.

Neither repeated tonography nor gonioscopy had any observable influence on the convalescence of any eyes included in this study.

Conclusion

Following lens extraction facility of outflow may be temporarily decreased, unchanged, or increased; usually the decrease is present early, in the period 3 to 12 weeks after surgery, but sometimes the decrease does not appear until late, 12 to 20 weeks after surgery. Those eyes with transient decrease often have a definitely elevated tension during the convalescent period, but sometimes they do not. Most such eyes do not require antiglaucoma therapy, but they should be watched carefully. When outflow facility is found to be increased, the wound is apt to contain incarcerated iris.

The facility of outflow and intraocular pressure of eyes with so-called exfoliation of the lens capsule, glaucoma capsulare, or open-angle glaucoma are essentially unchanged by cataract extraction.

The inner scar from cataract section may occasionally be situated so far posteriorly as to invade the trabecular meshwork (ordinarily the inner scar is somewhat anterior to Schwalbe's line, when tract sutures are used). Such encroachment of inner scar on the meshwork does not impair outflow facility.

Pigment dust in the inferior angle may be seen gonioscopically in a large number of cataractous eyes whose outflow is normal. After lens extraction, additional pigment dust may settle on the meshwork, with

concomitant lowering of C. Some of this pigment may later disappear, and C may return to normal.

In managing eyes with flat anterior chamber after cataract extraction, early gonioscopic examination should be done after the anterior chamber is reformed, in order to determine the presence and extent of peripheral anterior synechias; in some instances these synechias may be broken by the use of miotics.

Dr. W. Morton Grant and Dr. Paul A. Chandler gave criticism in the preparation of this paper. Dr. David D. Donaldson and his assistants, of the Howe Laboratory of Ophthalmology, prepared the goniophotographs.

243 Charles St. (14).

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Contact Lenses in Monocular Aphakia

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Two years ago[1] I discussed the present subject before the Section on Ophthalmology of the American Medical Association. I bring it up again because, in view of what is known about the method and the number of articles in the literature concerning it, too few ophthalmic surgeons are adopting it.

The first article I have found in the literature describing the use of a contact lens in monocular aphakia is that of Little,[2] who in 1934 removed a complicated cataract from a 17-year-old boy and obtained fusion with a contact lens. Town,[3] in 1939, reported three cases. Gettes and Ravdin[4] reported one case in 1949. Constantine and McLean,[5] in 1954, reported a series of 39 cases, the largest number recorded at that time. Since that time Gyorffy[6] has reported 25 cases and Levitt,[7] one. Levitt's patient was a 7-year-old boy, the youngest I know of who has successfully worn the lens. There may have been other reports that I have overlooked.

It is puzzling why more ophthalmic surgeons have not used this method of obtaining binocular vision in view of these reports on its value. Most ophthalmologists are conservative by nature and are slow to adopt a technique in which they have had no experience. The difficulty in finding a good contact lens fitter has no doubt deterred others. Any skilled optician can learn how to fit contact lenses, and, now that contact lens manufacturers are teaching the fitting technique, this should no longer be much of a problem. It is with the belief that more ophthalmic surgeons should adopt this method of managing monocular cataracts that I dare to present this subject after two more years of experience.

From Department of Ophthalmology, Baylor University College of Medicine.

It is necessary to recall what happens to an eye which develops cataract while vision remains good in the opposite eye, when the treatment is used that I shall call the "old method." This "old method" has been in vogue since Daviel removed the first cataract. Briefly, it consists in doing nothing as long as the patient can see very well with the other eye. Most surgeons choose 20/70 in the better eye as the criterion. Meantime, the first eye affected usually has lost the power of convergence, then has become exotropic. This accounts for the statement made by one prominent ophthalmologist[8] that surgeon and patient are happier when only one eye is operated on, as binocular single vision is seldom attained. This statement is correct if the first eye involved has become exotropic, as it is extremely difficult and often impossible to regain fusion once it has been lost because of a blind deviating eye. It is possible to keep such an eye from diverging by teaching the patient to overconverge by finger-to-nose exercise. In patients that refuse operation or delay it, this simple exercise, faithfully performed, will keep the eye in line for an indefinite period.

Every ophthalmologist knows that a person who has had one cataract removed, while vision in the other eye is good, cannot obtain binocular single vision when both eyes are corrected by spectacles. This is largely due to the difference in size of images. When the cataract lens is placed at the anterior focal plane of the eye, the image is about 30% larger than that of an emmetropic eye. Linkz,[5] working with Constantine and McLean, measured size images on the eikonometer in aphakics wearing a contact lens and found a size increase of 7.5%. This is easily within the bounds

of fusion capability. Patients wearing the regular cataract spectacle lens, with a thinner lens before the fellow eye, will seldom wear them except by compulsion, as from fear of losing a job, and they must alternate fixation and suppress the image seen by the other eye. I see no benefit whatever to this sort of procedure. Most such patients take their glasses off when they are not under scrutiny.

Another unfortunate complication frequently occurs if a cataract is allowed to go to hypermaturity, while one is awaiting the drop of vision in the other eye. This is secondary—or lens-induced—glaucoma. This comes about by toxic lens material seeping through the thin anterior or posterior capsule, producing uveitis and blocking the angle with macrophages. This undesirable complication has been encountered many times by every experienced ophthalmic surgeon, and, though it can often be managed successfully, it is easier to prevent than cure.

Mr. Harold Ridley, of London, attempted to solve the one-eyed cataract situation by an acrylic implant. Since his pioneering efforts other implants have been devised, but they are all based on a similar principle—placing an inert, foreign body within the eye. That it is not inert is proven by the fact that intraocular inflammation must be combatted from the start and that this often results in lowered vision and other complications. As I have had no personal experience with these implants, I will leave criticism to those who have tried this method and abandoned it and praise to those who have continued its use. To me it seems more sensible to place the foreign body outside the eye, where it can be removed at will.

Since my last report I have had 28 cases of monocular aphakia fitted with contact lenses, making a total of 63. Four of these patients have abandoned use of the lens for various reasons. One got the lens stuck on the cornea and could not remove it, and so it stayed in over night. This caused a corneal abrasion, which soon healed, but the patient refused to use it further. Another had early band keratophy, and the lens caused the calcium to flake off, keeping the eye irritated. A third wore the lens comfortably six months; then he apparently became allergic to methacrylate and gave up the lens in favor of spectacles. A fourth suffered detached retina and following scleral shortening has not secured enough vision to justify resuming the use of the contact lens.

Thirteen of the series have come to operation on the second eye. Three of these gave up the use of contact lenses in favor of spectacles; ten have continued to use them binocularly. The latter were all among the younger and more vigorous, who enjoy the wide field of vision, especially for driving. Several persons with bilateral aphakia who have never worn contact lenses have asked to be fitted with them to increase their field of vision for driving. Two of this series had lost the opposite eye and wear the contact lens because of the increased visual field.

It is necessary to review the procedure in selecting cases for operation and use of the contact lens. If the vision in the cataractous eye is 20/100 or less and reading vision is no longer present, it is time to operate. The vision in the opposite eye must be normal or nearly so, with slight or no evidence of lens opacities. The patient must not be feeble, clumsy, or highly nervous and should be still active, and he should be one in whom binocular vision is highly desirable. Also, the patient should be cooperative and intelligent enough to understand what we wish to accomplish. The situation must be explained to the patient so that he thoroughly understands what will be expected of him.

Eight or ten weeks after the operation, when the eye is white and the wound completely healed, the patient is tested for manifest refraction. If his vision is not 20/30 or better there is no use fitting the contact lens. If vision is approximately

normal with the lens, the vertex distance should be measured and the prescription sent to the contact lens technician. When the patient has been able to wear the lens four or five hours comfortably he should return to the surgeon with the lens in place. The surgeon determines the vision and examines the eye with the biomicroscope to see that the lens stays in place and yet floats enough to allow circulation of tears. The lens is then removed, and the cornea is stained with fluorescein. A little lacy staining of the cornea after five or six hours does not seem to matter. However, if the corneal epithelium is edematous enough to blur the vision, the lens is too tight for the proper circulation of tears and it must be reground or replaced. The patient is now tested for muscle imbalance. If he has primary fusion by the Worth dot test there will be no diplopia. If he cannot do this test properly he may have annoying double vision. Occasionally it is necessary to have prisms ground into the spectacle lenses, especially in vertical deviations. However, these deviations tend to disappear in time and prisms are seldom necessary. The degree of fusion does not seem to matter much. Some obtain primary fusion only. Many acquire third-degree fusion. There were three patients in this series who could not fuse for distance. However, they elected to wear the lens for the field of vision, learning to suppress enough to escape diplopia. One of these three, a dentist, can fuse for near and wears the lens while he works, reporting that he can see details so much better that he cannot work without it.

There are certain disadvantages to the use of contact lenses in aphakia that should be pointed out. Very few ophthalmologists have the time or the knowledge to fit contact lenses properly, and the others must depend upon a technician to do it for them. Many smaller cities and towns do not have a good contact lens fitter. The corneal lens and the microlenses are small and are easily lost. Insurance against this is cheap, and they can be replaced at little expense to the patient. The initial cost to the patient—about $100 per lens—is rather high, and this precludes a certain number from wearing them. The Veteran's Administration will not furnish contact lenses to patients operated on in their hospitals. These contact lenses sometimes need to be refitted after a few months, as the cornea seems to flatten somewhat when they are worn constantly. The only other disadvantage is to the ophthalmologists. These patients need more attention than those who wear correcting spectacles, and they usually drop in without warning.

In my opinion, the advantages of this method of treating persons with monocular aphakia far outweigh the disadvantages. An eye is put to work that was formerly useless, and binocularity is obtained in a very high per cent of properly selected cases. The problem of exotropia and the danger of lens-induced glaucoma are dissipated. The patient is left in the happy state of mind in which he knows that he will not be blind if some accident should happen to the other eye.

Summary

The results of the use of the contact lens in monocular aphakia in 63 patients are reported. Four patients abandoned the lens for various reasons. Thirteen patients have come to operation on the second eye. Three of these wear the classical cataract spectacles; ten wear contact lenses binocularly.

This method of treating monocular cataracts is not being used as much as its success justifies.

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Keyhole and Peripheral Iridectomies in Different Eyes in the Same Patient

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Whether an ophthalmologist prefers a broad basal iridectomy (keyhole) or the peripheral iridectomy or iridectomies (round-pupil) depends in a large part upon where he is practicing and where he received his training. However, a keyhole iridectomy is usually the choice in eyes where an old uveitis exists or in those cases where pupillary dilatation is poor. There is another category in which a broad basal iridectomy is considered essential by some ophthalmologists. That is, in cases where the first eye developed a retinal detachment or pupillary block glaucoma from vitreous herniation.

The reasons for maintaining the round pupil are, as we all know, as follows:

1. There is a more perfect optical system, said by some to be more physiologic, with resulting (*a*) less glare, (*b*) less astigmatism, (*c*) better depth of field.
2. It is cosmetically more pleasing.
3. There is less chance of vitreous loss.
4. There is more effective use of miotics in glaucoma.
5. The iris keeps the vitreous in its proper place and prevents the hyaloid from rupturing.

To elucidate the answer to this important problem, we feel that the best controlled experiment is to find uncomplicated cataract extractions in humans in which one eye had a peripheral iridectomy or iridectomies (i. e., a round-pupil extraction) and the other, a broad basal iridectomy (keyhole extraction). There were many cases to be found, both at the Massachusetts Eye and Ear Infirmary and in Detroit, in which the first eye had a round-pupil cataract extraction followed by a broad basal iridectomy in the other eye. However, almost always the second type of operation was felt necessary because of serious complications in the first, i. e., glaucoma, vitreous loss, retinal detachment, connective tissue infiltration, epithelial downgrowth, prolonged and severe uveitis, or drawn-up pupil. Naturally, it would not be a fair evaluation to include these cases. We are therefore limiting our series only to those cases in which the operative and postoperative course was uneventful except for slight hyphema or choroidal detachments which rapidly and completely cleared, leaving no residual damage. We have examined all of these patients to corroborate what has been found. After observing that there was no radical difference between the two eyes, we have performed this procedure on several of our own private and ward patients.

Report of Cases

In our series we have discarded no uncomplicated case which had a round pupil in one eye and a keyhole in the other. We are first presenting three cases in which bilateral fundal pathology was present, which could not be recognized until the mature cataracts had been removed from both eyes.

CASE 1.—A 71-year-old widow, on June 28, 1955, had an uneventful combined cataract extraction O. S. It was noted that she had gyrate atrophy, and it was decided to remove the cataract in the other eye. The surgeon performed a round-pupil extraction, on July 1, 1955, both uneventful. Postoperatively, there were slight hyphema in both eyes lasting two to three days, occurring on the fourth postoperative day. Both fundi showed the same degree of gyrate atrophy. Vision was O. S. $+1200 -50\times90=3/200$ (peripheral iridectomy); $+1300-200\times90=5/200$ (combined). The

patient could tell no difference in glare or distortion in her vision.

CASE 2.—A 74-year-old married woman, much younger than her years, had round pupil extraction O. S. on June 10, 1955, and a combined extraction O. D. on June 14, 1955. Patient's postoperative course was uneventful. On Aug. 19, 1955, O. D. $+1350-200\times50=20/50$ (keyhole); O. S. $+1400-250\times75=20/50+2$ (round-pupil); +250 add gives 62 M. The patient had pigmentary macular disturbance typical of early senile degeneration. She had no glare or distortion; neither did she notice any difference between her eyes.

CASE 3.—A 69-year-old bachelor, much younger than his years, on Oct. 25, 1954, O. D., had a combined and on July 12, 1955, O. S., had a round-pupil cataract extraction. Postoperatively there were no problems. Vision was O. D. $+850-150\times75=20/40+2$ (keyhole); O. S. $+800=20/30-1$ (round-pupil); +250 add gives 62 M. This patient noticed no difference in distortion or glare between his two eyes. Both fundi showed moderate peripupillary atrophy.

The remaining cases are those in which there were no demonstrable abnormalities in the fundus:

CASE 4.—The patient was an 84-year-old widower, appearing 15 years younger than his stated age, with 20/200 vision in both eyes. On July 27, 1956, the cataract O. S. was removed with a round-pupil extraction, and on Aug. 2, 1956, the cataract O. D. was removed with a large keyhole iridectomy, approximately the upper half of the iris. On Aug. 13, 1956, an air injection with posterior sclerotomy O. S. was performed because of an injected eye with a flat chamber and large choroidal separation. Postoperatively and ever since, when the sun has been out on the snow, he has noticed that there is a moderate amount of glare in the right eye in comparison with the left. On Jan. 23, 1957, O. D. $+1275-300\times180=20/25+2$ (combined); O. S. $+1225-250\times70=20/20+1$ (round-pupil); $+250-37$ M.

CASE 5.—The patient was a 64-year-old widower appearing much older than his stated age, with preoperative vision O. D. 15/200 and O. S. acc L. P. On Sept. 2, 1955, a combined O. S. and on Sept. 13, 1955, a round-pupil extraction O. D. were performed. On the sixth postoperative day, there was a slight hyphema O. D. On Dec. 1, 1955, O. D. $+1100-250\times85=20/20$ (round-pupil); O. S. $+1100-350\times90=20/30+1$ (keyhole). The patient was unable to tell any difference between the two eyes.

CASE 6.—A 53-year-old white housewife had preoperative vision of 20/300 O. S. and 20/70 O. D. On Oct. 26, 1955, a round-pupil extraction was performed O. S., and then on Sept. 7, 1956, a combined extraction O. D. She experienced no difficulties postoperatively. On Nov. 20, 1957, O. D. $+1250+100\times145=20/30$ (keyhole); O. S. $+1275+100\times43=20/25-2$ (round-pupil); +250 add 37 M.

CASE 7.—A young-appearing 67-year-old white man had vision O. D. 15/70 and O. S. 15/200. On June 28, 1955, a keyhole iridectomy O. S. and on July 1, 1955, a combined extraction O. D. were performed. His postoperative course was uneventful, and except for diplopia from a muscle imbalance, which was corrected by prisms, the patient could tell no difference between the two eyes. In June, 1956, O. D. $+1000+150\times180=20/20+$ (round-pupil); O. S. $1000=20/20+$ (keyhole); 4Δ base-out O. U.; +250 add 37 M.

CASE 8.—A 45-year-old ex-truck-driver showed 15/100 vision in both eyes preoperatively. On June 7, 1955, O. S. had a round pupil extraction, and on June 10, 1955, O. D. had a combined extraction. Postoperatively there were no difficulties, and the patient returned to his occupation, never noticing any difference between his eyes. On Aug. 8, 1955, O. D. $+1150-275\times60=20/20$ (keyhole); O. S. $+1200-200\times85=20/20$ (round-pupil); +250 add 37 M.

CASE 9.—A practicing registered nurse who works almost exclusively with eye pa-

tients had preoperative vision of 1/60 O. D. and 2/60 O. S. In November, 1946, she had a combined extraction and in July, 1948, she had a round-pupil extraction. She is unable to notice any difference between her eyes. Vision is +1450−100×105=6/5 (keyhole); +1450−100×75=6/5 (round-pupil); +300 add.

CASE 10.—A 60-year-old white man on admission had 20/200 vision O. U. On Dec. 24, 1954, O. D. had a keyhole iridectomy, and on Dec. 28, 1954, a combined extraction was done. The postoperative course was uneventful except for inability of the left pupil to dilate. The patient has never noticed any difference between the two eyes. Vision is O. D. +1000−50×80 =20/20 (keyhole); O. S. +1050=20/25 (round-pupil); +250 add 37 M.

CASE 11.—A belligerent bartender on admission showed vision of 20/100 O. D. and accurate light perception O. S. On May 23, 1955, a round-pupil extraction was done, followed by a combined extraction on May 27, 1955. The postoperative course was uneventful except that O. D. was bothered by the light for the first few weeks; after this there was no difference between the eyes. On Aug. 2, 1955, O. D. +950=20/20 (keyhole); O. S. +950−150×75=20/25 (round-pupil).

CASE 12.—A young 45-year-old father of three was admitted preoperatively with 20/200 vision O. D. and 20/100 vision O. S. On June 28, 1947, a round-pupil extraction was performed, followed by a combined extraction O. S. Both eyes showed slight choroidal detachments, which cleared nicely, and the patient was unable to distinguish any difference between his eyes. Vision was O. D. +1300=20/25 (round-pupil); O. S. +1500−250×65= 20/20 (keyhole); +250 add 37 M.

CASE 13.—A 67-year-old retired shoe salesman had preoperative vision of 15/70 O. D. and 15/50 O. S. On June 24, 1955, a round-pupil extraction was performed O. D., followed by a combined extraction O. S. There were no complications and no noticeable difference between the two eyes. On Oct. 11, 1955, O. D. +1275− 125×80=20/15−2 (round-pupil); O. S. +1175−100×100=20/15 (keyhole); +250 add 37 M.

CASE 14.—A 64-year-old tool and die maker had preoperative vision of O. D. 20/50 and O. S. 20/100. On July 7, 1956, a keyhole extraction was done O. S., followed by a round-pupil extraction O. D. Postoperative course was uncomplicated. The patient noticed that glare from the snow bothers his right eye more than his left eye. On April 1, 1957, O. D. +1300 −200×100=20/40+1 (round-pupil); O. S. +1300−175×88=20/25+2 (keyhole); +250 add O. D. 75 M; +250 add O. S. 37 M.

Summary of Cases

Case	Preoperative Vision		Postoperative Refraction		Postoperative Vision		Difference Between Eyes
	Keyhole Extraction	Round-Pupil Extraction	Keyhole Extraction	Round-Pupil Extraction	Keyhole Extraction	Round-Pupil Extraction	
1	L.P.	L.P.	+1300—200×70	+1200— 50×70	5/200	3/200	None
2	L.P. & H.M.	L.P. & H.M.	+1350—200×50	+1400—200×75	20/50	20/50+2	None
3	L.P. & H.M.	L.P. & H.M.	+ 850—150×75 normal fundi	+ 800	20/40+2	20/30—1	None
4	20/200	20/200	+1275—300×180	+1225—250×70	20/25+2	20/20+1	Glare greater in keyhole
5	L.P.	25/200	+1100—350×70	+1100—250×85	20/30+1	20/20	None
6	20/70	20/300	+1250+100×145	+1275+100×43	20/30	20/25—2	None
7	15/200	15/70	+1000	+1000+150×180	20/20+	20/20+	None
8	15/100	15/100	+1150—275×60	+1200—200×85	20/20	20/20	None
9	1/60	2/60	+1450—100×105	+1450—100×75	6/5	6/5	None
10	20/200	20/200	+1000— 50×80	+1050	20/20	20/25	None
11	20/100	L.P.	+ 950	+ 950—150×75	20/20	20/25	None
12	20/100	20/200	+1500—250×65	+1300	20/20	20/25	None
13	15/50	15/70	+1175—100×100	+1275—125×80	20/15	20/15—2	None
14	20/100	20/50	+1300—175×88	+1300—200×100	20/25+2	20/40+1	Glare greater in round pupil

Comment

The excellent papers of Post and Harper,[1] Knapp,[2] Owens,[3] Hughes and Owens,[4] Davis,[5] and others comparing their results with round-pupil extractions versus broad basal iridectomy certainly leave no room for argument in that round-pupil extraction was by far the safer method at the time these papers were written. The incidence of vitreous loss is approximately four times greater with the combined method, and there certainly is a significantly better visual result in all these series. However, with the advent of hyaluronidase in lidocaine (Xylocaine) and other long-acting local anesthetics, giving digital pressure on the globe after retrobular injection to reduce intraocular pressure, speculums or lid sutures which do not press on the globe, multiple preplaced cornea-scleral sutures, and good preoperative sedation, it is not unusual for busy ophthalmic surgeons to speak not in days, but in years concerning the last time they lost vitreous when using the combined technique. We know of one resident at the Massachusetts Eye and Ear Infirmary who went through his entire residency, during which he performed more than 130 cataract extractions, without once losing vitreous.

In the cases which we are here presenting there are, as previously stated, no complications present in either eye, and so miotic function could not be enlisted in the case of postoperative glaucoma. It is of interest that only two patients noticed any difference in glare. One of the two men (Case 4), had an iridectomy that was approximately the upper half of the iris; in his other eye was a 2 mm. pupil which could not be dilated. He certainly fits into the category of Dr. Berliner,[6] "This round pupil eye feels and sees better." The other case (Case 14), however, is one in which the glare, surprisingly enough, was greater in the eye with round-pupil extraction.

Upon looking over the cases, except for three, we have found a slightly greater astigmatic error (0.25 to 0.75 D.) in the keyhole eye. The average astigmatism for the keyhole extraction was 1.61 D., and, for the round-pupil extraction, 1.3 D. However, in the cases with the normal fundi the visual acuity has been better, five to three cases, in the keyhole eye. One of the patients with peripheral iridectomy, for unexplained reason, had a much poorer result, the other three having equal vision. There was no noticeable subjective difference as far as depth of field was concerned between the two eyes, but only questions and rough tests were employed, i. e., such as difference between two pencils.

The drooping lids of advancing years are a great aid to the ophthalmologist who does perform keyhole iridectomies and who is concerned with the cosmetic appearance of his patients. In all these patients the lids very nicely completed the missing portion of the pupillary sphincter. We have also observed in our younger patients in whom keyhole iridectomies were performed that the lids assumed this same position after surgery.

All the eyes were examined to see whether there was any difference in the hyaloid faces. In all these eyes it was intact, and there did not seem to be any difference in amount of herniation in fellow eyes.

Since a large part of the practice of one of us (D. S. J.) is the care of retinal detachments, the hours spent looking for the retinal holes through an undilatable pupil in a tense, frightened person, whose globes are constantly wiggling about, make him very grateful to the surgeon who has performed the keyhole iridectomy. Since an eye that has had its lens removed usually has other degenerative changes, one is no longer dealing with a physiologic eye, and, since it does not seem to make an appreciable difference in the visual outcome of the relatively normal or grossly abnormal eyes presented here, we should like to quote from a paper written by Dr. L. F. Carter[7]: "otherwise perhaps [round pupil is] more often a delight to the surgeon than to the patient."

Summary

Fourteen cases of bilateral aphakia, one eye having a round-pupil, the other eye, a keyhole iridectomy, are presented. Three of these cases had equal fundal changes. The other 11 had normal fundi.

There was no noticeable subjective difference between the two eyes except in two cases. In one case the round-pupil eye noticed greater glare, and in the other there was greater glare in the eye with the keyhole iridectomy.

Hyaloid faces in all these cases were intact, and there did not appear to be any differences in bulge into the anterior chamber.

The visual results were slightly better in the eyes with the combined extraction.

The lids covered the broad basal iridectomies, making them cosmetically indistinguishable from the round-pupil extractions.

There was slightly greater astigmatism in the keyhole extractions (1.6 to 1.31 d.).

The Board of Surgeons of the Massachusetts Eye and Ear Infirmary, and Drs. E. B. Dunphy, Paul Chandler, David Cogan, Josiah Smith, S. J. Bullington, D. F. Milam, Maurice Hauser, Saul Sugar, and Sydney Stone allowed us to use their cases and gave help in preparing the manuscript.

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Conservatism in the Management of Congenital Cataract

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In our experience, the treatment of congenital cataract has not been satisfying. That this feeling is not unique with us is borne out in a number of excellent publications during the past several years. The authors of these almost uniformly point out the difficulties and complications in such treatment and urge conservatism.

It is our purpose in this paper to outline a conservative approach to the management of congenital cataract and to document the suggested management by citing appropriate cases.

I. References to the Literature

Some review of the literature is illuminating. Falls,[1] in reporting 233 eyes operated on, noted that only 35.5% obtained a vision of 6/12 or better, while 25.6% had a vision of 6/60 or less and at least 6.4% were blind. He noted that other ocular and systemic defects are often associated with congenital cataract, and such association makes the prognosis for successful treatment very guarded. Owens and Hughes[2] further emphasized the association of other ocular defects with congenital cataract and noted the poor final vision resulting from treatment. They stated that only 11% of these eyes obtained a vision of 20/70 or better, while 42% had a vision of less than 20/200. They also stated that visual acuity resulting from surgery after the age of 2½ years was definitely better than when surgery was performed earlier, but they recognized that the more grossly defective eyes would naturally be operated on earlier, and the prognosis would be much poorer than in the less defective eyes, whose surgery could be reasonably deferred to a later age. They specifically stated that no eyes having associated defects and operated on before the age of 2½ years obtained a vision of 20/70. An interesting additional comment, not supported by some other writers, is that complications were no more frequent in congenital than in senile cataract surgery and were of about the same incidence in congenital cataract whether it was associated with other ocular defects or not.

Bagley,[3] in reviewing a later series of congenital cataracts from the Wilmer Institute, notes that only 36.8% of his series showed gross associated defects, as opposed to 55.8% cited by Owens and Hughes. He comments that in the later series ". . . operations have not been performed on many eyes having gross defects, which would have been subjected to operation in the past." Bagley also noted that in his series a smaller percentage of patients were operated on before the age of 4 years than were operated on before the age of 2½ years in the Owens and Hughes series. These two facts bespeak a recognition of the need for conservatism in treating congenital cataract.

Chandler[4] and Cordes,[5] in 1951, gave indications for surgical intervention in congenital cataract, recognizing that total or large dense central opacities necessitate surgery early in life. Cordes states that "when there are bilateral dense central opacities and useful vision can be obtained only by dilating the pupils, surgical intervention is always indicated." Chandler feels that dilatation may be helpful but that surgery of the lens eventually is necessary. We will speak of this later. Chandler makes the interesting and pertinent statement that in bilateral moderate involvement "ambly-

opia does not develop in these cases, and we should wait until the child is 5, 6 or 7 years of age so that an accurate refraction can be done, and one can be absolutely sure that corrected vision is below the critical level." Again, to quote Chandler, ". . . if operation is done in one eye, that eye is certain to become amblyopic." We agree with both of these thoughts.

Most writers have emphasized the complications of the surgery of congenital cataract, mentioning specifically glaucoma, vitreous loss, membrane formation, updrawn pupil, occlusion of the pupil, anterior peripheral synechiae, and retinal detachment. Cordes [6] recently enlarged on all of these while discussing failure in congenital cataract surgery. Chandler,[4] in urging conservatism, says "the first point to be considered in avoiding retinal detachment in these cases of congenital cataract is not to operate on the patient in the first place." This same admonition may well apply to all of the other complications of lens surgery.

Little comment appears in the recent literature regarding the treatment of monocular congenital cataract, though occasional sources [4,7] state that surgery is not helpful and is often harmful. Cases of monocular congenital cataract surgery with production of satisfactory visual acuity have not come to our attention.

While many writers mention optical iridectomy as a possible method of treating congenital cataract, only Chandler [4] recently has been favorably impressed with the use of this procedure in properly qualified cases. He cites one case. We are impressed by the scarcity of such reports and also by the infrequent reporting of the prolonged use of mydriatics in the treatment of congenital cataracts.

II. Personal Experience and Illustrative Cases

It is our purpose to emphasize the use of mydriatics and optical iridectomy in certain groups of cataract and to deplore the use of surgery for monocular congenital cataract.

The following thoughts and impressions have been gained from a review of 100 cases of congenital cataract, followed over a period of years in our private practice. We recognize at least five degrees of severity of involvement of the lens in congenital cataract. They are (1) bilateral minimal involvement, (2) unilateral cataract, (3) bilateral moderate lamellar involvement, (4) bilateral dense nuclear involvement with clear cortex, and (5) bilateral extensive involvement.

GROUP 1.—*Bilateral Minimal Involvement*

This is, fortunately, the commonest type of congenital cataract. It includes the polar and particulate types of lens opacification. Visual acuity is affected very little, and the patient suffers no handicap. An adequate refraction should be done, and the influence of mydriasis should be estimated. Treatment is usually not necessary.

GROUP 2.—*Unilateral Cataract*

This group of cases, with severe lens involvement in one eye and slight in the other, requires some thought and consideration. The patient's presenting complaint is strabismus, unilateral microphthalmos, and/or photophobia, with a marked tendency to close one eye in the bright light.

In our series of 20 cases strabismus was the chief complaint at the time of the first visit. At the time of this review, every case had an evident strabismus (frequently with a large vertical element), and strabismus surgery had been performed on eight for cosmetic reasons. Further, seven cases complained primarily of very annoying photophobia and a tendency to close the affected eye in the light.

It is important to note that surgery of the affected lens (three cases operated on by us, and five, by others) did not lessen the photophobia, and in no case did it favorably influence the strabismus. Of even greater importance is the fact that in no case was the vision of the affected eye better than the recognition of hand movements, whether surgery had been performed or not. In no

operated-on case were we able, with occlusion and glasses, to improve the visual acuity, in spite of good cooperation on the part of the patient and parents.

A 3-year-old boy had been operated on for cataract O. D. at the age of 1 year, which resulted in a clear slightly irregular pupil. O. S. was normal. At the age of 3 years he had pendular searching nystagmus O. D. He wore correction O. D. and constant occlusion O. S. for six months, with no visual improvement. Final examination revealed (1) vision O. D. with +12.00 sph=5/200, roving nystagmus, and vision O. S. with +1.25 sph+0.75 cyl×85= 20/30+; (2) pupil O. D. somewhat small and sluggish; (3) variable esotropia. No further treatment was recommended except possible strabismus surgery in the future.

Thus, since visual acuity is not improved, strabismus is not favorably influenced, and photophobia is not alleviated, we would unequivocally advise against surgery in unilateral congenital cataract unless the cataract is becoming hypermature. The appearance of the eye is usually not improved; function has not been helped, and the eye has been jeopardized if surgery is performed.

GROUP 3.—*Bilateral Moderate Lamellar Involvement*

This group, consisting of eight cases, offers a fertile field for careful conservative clinical judgment. The patient, having a moderate lamellar involvement of both lenses, often may be able to obtain 20/50 or better vision in subdued light or when the pupils are dilated by a mydriatic but sees poorly when the pupils are contracted by bright light or when reading. Even in the child too young to read the Snellen chart, it may be evident that visual "fixation" is central and secure when the pupil is dilated (thus exposing clear cortex) but insecure when the pupil is normally constricted.

The patient who demonstrates satisfactory improvement in visual acuity on mydriasis should have his eyes kept dilated with atropine or homatropine daily; refraction should be done as carefully as possible, and the proper bifocals should be prescribed. As the patient approaches the school age, if visual acuity, when the eyes are dilated, is 20/50 or better, either he should have them kept dilated and wear the proper bifocals or optical iridectomy should be performed. While an optical iridectomy causes some dazzling and some visual distortion because the light rays are passing through the periphery of the lens and the cornea, the minimal hazard of the surgery done, the obvious advantage of keeping the accommodation active, and the fact that a heavy aphakic correction is unnecessary far outweigh the disadvantages. We have operated on two such patients, with obviously happy results. One of these cases is reported here.

A 10-year-old boy complained of dazzling and not seeing to read well. Examination revealed active pupils of 3 to 5 mm. in subdued light; a moderate lamellar cataract was noted in each eye. Normal fundi could be seen through the clear lens periphery. In brighter light, the lens opacity almost filled the pupillary space. Vision O. D. was 20/70; with mydriatic, 20/40+. Vision O. S. was 20/50; with mydriatic, 20/30. Vision O. U. at 13 in., J 8; with mydriatic and correcting lens, J 3+.

The eyes were kept dilated for eight years, with use of homatropine 5% b. i. d. at first, but later drops were needed four times a day. Tinted bifocals were worn. School and daily activities were fair except for the handicap of dazzling and the lack of accommodation.

At the age of 19 years the lens opacities had not changed. Optical iridectomies were done at 5 and 7 o'clock in right and left eyes, respectively, without incident. Two months later the patient had a visual acuity of 20/25 in each eye at 20 ft. without correction, and a vision of 20/30— in each eye at 13 in., also without correction.

The visual acuity continues to be good for 20 ft. and 13 in. one year after operation. Visual distortion is minimal, and

dazzling is only slightly annoying. No mydriatic has been necessary for college studies or normal activities since the iridectomies were performed.

These patients whose eyes have been kept dilated and who, on approaching school age, do not have adequate vision, should be operated on, probably with performance of a linear extraction. If good central fixation has continued to be present, there have been several advantages to delaying the surgery: 1. A more accurate estimate of visual acuity has been possible, and the necessity for surgery has been firmly established. 2. Severe amblyopia has not developed in one eye because of early surgery on one eye and none on the other. 3. Binocular single vision has usually developed, and the subsequent onset of strabismus is much less likely. 4. The patient, being older, is more cooperative during the time of surgery and the postoperative period. 5. The eye is more mature; the anterior chamber is deeper, and the pupil is easier to keep dilated.

GROUP 4.—*Bilateral Dense Nuclear Involvement with Clear Cortex*

This group is composed of 19 cases. These are often seen associated with other anomalies such as microphthalmos, shallow anterior chamber, small resistant pupil, nystagmus, and strabismus. Nine cases gave a definite history of maternal rubella in the first trimester. The others were clinically similar but with uncomplicated pregnancy, and we have termed them "rubella-like" cataract. Our experience with this group has been most unhappy, and our final visual results, poor. Of 34 eyes operated on, only one obtained a vision of 20/70, and only three, a vision of 20/200 or better, while six eyes were totally blind from glaucoma or opacification of the cornea.

Since these pupils will not dilate and stay dilated well, some surgery seems important. Some years ago we performed discission upon these cases; later iridectomy and linear extraction was preferred, and in two recent cases these were accompanied by sphincterotomy below. In spite of these operative attempts, the surgery has continued to be difficult, and the end-results, not satisfactory.

With this in mind, more recently we have done complete iridectomies at 12 o'clock in one such case, with definite improvement of the apparent visual acuity and with preservation of the integrity of the globes. At some later time, when visual acuity can be determined more accurately, lens surgery may be considered wise, but not at present, in our judgment.

A 3-month-old boy had had nystagmus since birth, and a diagnosis of cataract was made. Examination revealed an alert baby with marked ocular nystagmus. Undilated, the pupils were 3 mm., and no clear lens was evident. Vision was limited to vague following movements. With mydriatic, the pupils dilated to 5 to 6 mm. and dense nuclear opacity with clear periphery was evident in each eye. Visual alertness was improved with mydriasis. He was treated by the use of homatropine 5% t. i. d. (he was sensitive to atropine) for the next four months.

At the age of 7 months, complete iridectomies were performed at 12 o'clock, without complications. At the age of 20 months, there is a clear zone of lens through the iris coloboma O. U. The patient seems to see poorly at a distance but well near. Nystagmus is slower and of less excursion.

Lens surgery may be performed in the future, though prognosis for good vision is uncertain.

GROUP 5.—*Bilateral Extensive Involvement*

This group, consisting of eight cases, is somewhat similar to Group 4, except that the lens involvement is more extensive, clear cortex is slight or absent, and associated anomalies are less frequently present. It would seem wise to perform surgery by the first year of life. Linear extraction with complete iridectomy is probably the operation of choice. The second eye may be operated on as soon as the outcome of the

first operation is evident. These patients respond to surgery better than those in Group 4. Further comment on this group is not of primary importance to this paper.

III. Summary and Conclusions

The literature and our experience have been cited to show the operative difficulties, the rather high incidence of complications, and the relatively poor visual improvement resulting from much of the surgery for congenital cataract.

Five groups of congenital cataract, based on severity of involvement, are discussed, and methods of managing each are suggested.

The limited but significant role of optical iridectomy and the prolonged use of mydriatics are pointed out, and the relative safety of these procedures is emphasized.

Finally, surgery for unilateral congenital cataract is strongly advised against.

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Studies of the A-Wave in the Human Electroretinogram

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The electroretinogram (ERG) in response to high-intensity stimuli represents a sequence of waves beginning with a negative deflection—the a-wave. Previous studies have analyzed this complex wave in terms of photopic and scotopic components. These analyses were derived from the behavior of the a-wave during dark adaptation and with stimuli of different wavelengths.[1-4] This assumption of a duplex nature of the a-wave has been strengthened by the fact that it frequently appears to be split into two parts separated by a "step," or hump. Based on this finding, tentative schemes have been suggested in which the photopic and scotopic components of the a-wave are distinguished by different latencies.[1,4]

The present study deals with the a-wave of normal, congenitally night-blind, and totally color-blind subjects in response to white-light stimuli of different intensities. Since the rapid course of the a-wave prevents adequate measurements when it is registered with standard ERG techniques, the speed of recording has been increased to demonstrate every detail of this wave, thus eliminating the later positive waves from the record.

Methods

The ERG potentials were picked up from the anesthetized cornea of the right eye with a Burian-Allen contact lens electrode. The reference electrode was applied to the bridge of the nose, and the ground electrode was placed against the forehead. The amplifier system consisted of a capacity-coupled preamplifier (Grass P-5) which was connected with one of the direct-coupled main amplifiers in a double-beam cathode-ray oscilloscope (ETC K 26). The frequency response of the system was flat between 1 and 10,000 cycles per second.

The light stimuli produced by a gas-discharge lamp (Grass Photic Stimulator PS 1) were flashes of about 10 microseconds' duration and a peak luminance of about 6×10^7 millilamberts (=100 arbitrary units). The stimulus patch had a diameter of 19 degrees and was equipped with a small red fixation light. Stimuli of longer duration were produced by a simple device consisting of an opalescent incandescent bulb (150 watts) and a photographic shutter (stimulus patch 14 degrees in diameter, luminance about 20,000 millilamberts). White-light stimuli were used exclusively. Neutral filters (Inconel) varied the light intensity.

Recordings were made with a Grass Kymograph Camera (Model C D); the single-frame operation was used in most experiments. A Grass Electric Stimulator (Model S 4 B), connected with the relay output of the camera, triggered the single sweep of the cathode-ray oscilloscope, and, after a set delay, the Photic Stimulator. In this way the entire stimulating and recording system was operated by the manual switch of the camera.

The light stimulus was recorded by a photocell and the second channel of the oscilloscope. At the lateral edges of the oscilloscope screen the beams were found to be slightly asynchronous. To obtain accurate time measurements, a stimulus artifact produced by the Photic Stimulator was

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superimposed on the ERG tracing. Its perfect synchronism with the light flash was verified by control experiments. In several experiments the ERG was recorded using the moving film operation (500 mm. per second), with the oscilloscope sweep switched off.

Records were obtained from five normal subjects with no ocular complaints or evidence of ocular pathology. A totally color-blind and a congenitally night-blind subject were also studied. Clinical and ERG findings in these two cases were reported in a previous publication.[8]

ERG records were obtained in complete darkness with a dilated pupil (phenylephrine [Neo-Synephrine] hydrochloride 10%). The recordings were begun after 30 minutes of dark-adaptation following a previous exposure to the average room illumination of 8 ft-c.

The mean values of about 20 responses were calculated for each subject at each intensity. In the individual subject the coefficients of variation were remarkably low for amplitudes and implicit times * (1% to 3%) but much higher for latencies (10% to 12%), owing to difficulties in measurement. All measurements were made with a microfilm reader.

The results are based on a total of 847 records, of which 571 were used for quantitative evaluation.

Results

Figure 1 shows the normal ERG elicited by intense light flashes and recorded with high speed to demonstrate details of the a-wave. With the very brief flashes produced by a gas-discharge lamp (Fig. 1*A*) the temporal pattern of response is fundamentally the same as with the light flashes produced by incandescent light and photographic shutter (Fig. 1*B*). In both cases the negative initial response is split into two parts (a_1 and a_2) separated by a "break." The latter appears as a positive hump (Fig. 1*A*) or merely as a "step" (Fig. 1*B*). For the purpose of this study, the "break" will be called Hump 1 (h_1) to distinguish it from Hump 2 (h_2) and Hump 3 (h_3) in

* Implicit time refers to the interval from onset of stimulus to peak of wave.

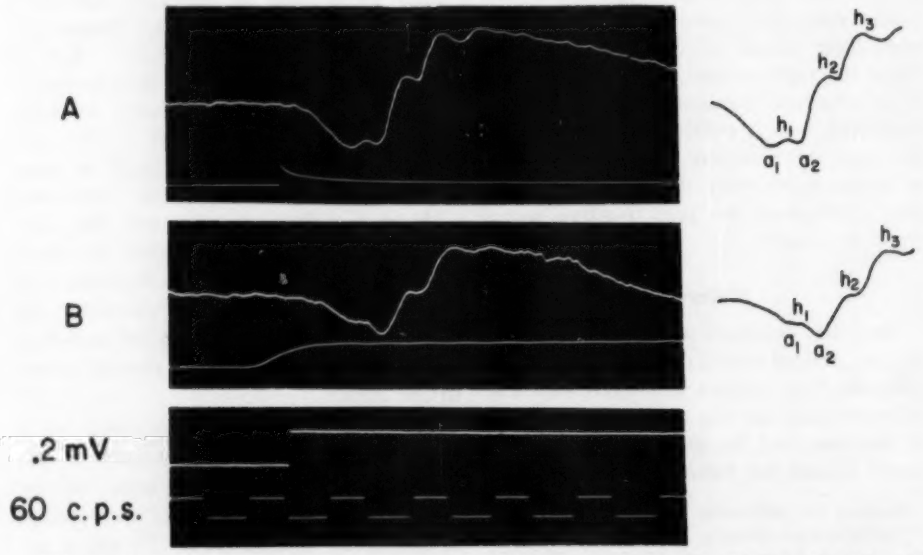

Fig. 1.—Normal human electroretinogram with high-intensity flashes of different duration. *A*, 10 microseconds (gas-discharge lamp). *B*, 100 milliseconds (incandescent light, photographic shutter). Right, labeling of waves for this study as suggested in text.

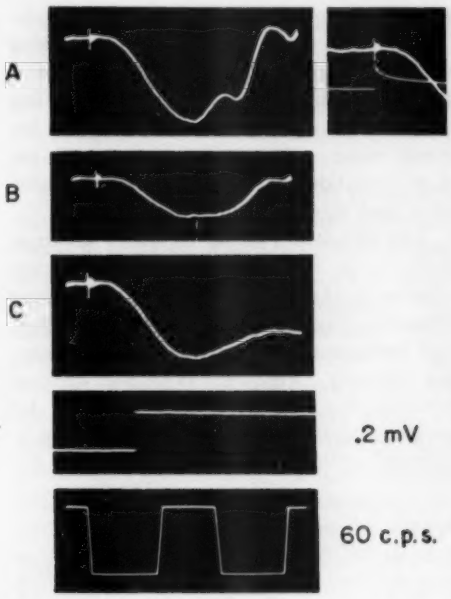

Fig. 2.—Original records of the a-wave in normal (*A*), totally color-blind (*B*), and congenitally night-blind (*C*) subjects. Gas-discharge lamp, intensity 100. Right upper corner, control recording showing synchronism of the stimulus artefact in the ERG and the light flash registered by a photocell.

the ascending limb of the following positive wave (see Fig. 1, labelled diagram).

The initial response was studied in detail with use of the gas-discharge lamp for stimulation. Figure 2 shows ERG's obtained with the brightest light flash in a normal, a totally color-blind, and a congenitally night-blind subject. The humps regularly found in normal subjects were absent or less pronounced in the color-blind subject and completely lacking in the night-blind subject. Most important was the finding that the latency of the a-wave in all cases was of the same order. This is illustrated by Figure 3, which shows the latencies of the a-wave found with four different stimulus intensities in the five normal controls and both subjects with retinal anomalies. The values of the latter two subjects fall clearly within the normal range.

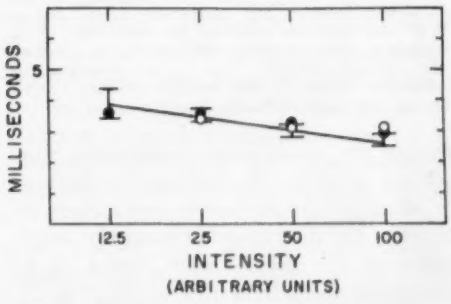

Fig. 3.—Latency of a-wave with different stimulus intensities (gas-discharge lamp). The curve and the vertical bars (I) represent mean values and standard deviations of five normal controls. Open circles, totally color-blind subject. Filled circles, congenitally night-blind subject.

The complex pattern of the normal initial response is shown in two typical examples (Fig. 4). With increasing stimulus intensity, not only do the implicit times of the

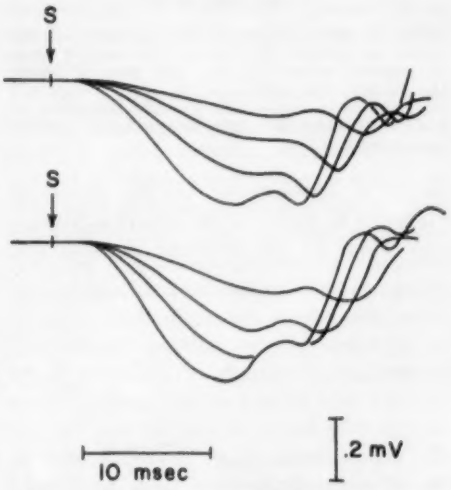

Fig. 4.—Pattern of a-wave and initial humps (see labelled diagram, Fig. 1) with four different stimulus intensities: 12.5, 25, 50, and 100 arbitrary units. Two normal subjects but differing in type of pattern ($a_1 > a_2$; $a_1 < a_2$). Light source was a gas-discharge lamp.

negative and positive peaks (a_1, a_2, h_1, h_2, and h_3) tend to become shorter but there is also a shift in the proportion of the amplitudes of a_1 and a_2. This proportion, furthermore, was found to vary considerably from subject to subject, as demonstrated in the two examples of Figure 4.

The influence of stimulus intensity on the constituents of the normal initial re-

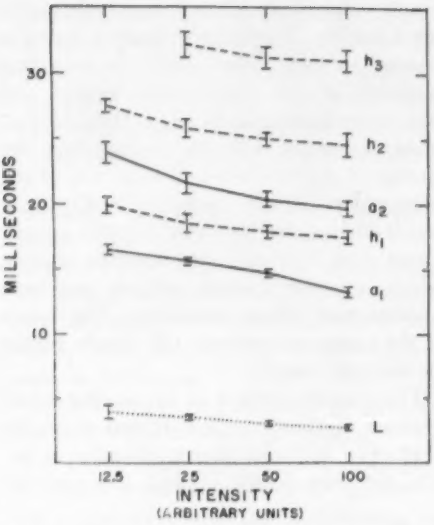

Fig. 5.—Influence of stimulus intensity on the latency of a-wave (L) and on the implicit times of negative waves (a_1, a_2) and positive humps (h_1, h_2, h_3). The curve and the vertical bars (I) represent mean values and standard deviations of five normal controls. Light source was a gas-discharge lamp.

sponse is demonstrated by the graphs in Figures 5, 6, and 7. With respect to implicit time (Fig. 5) the different negative and positive peaks behave in a similar manner: The implicit time decreases with increasing stimulus intensity. The small range of variation in implicit times observed with five different subjects is noteworthy. This becomes more evident in comparison with the wide variation revealed by the amplitudes of a_1 and a_2 (Fig. 6).

The amplitude of a_1 increases continuously with increasing stimulus intensity, whereas a_2 fails to increase at the highest intensity (Fig. 6). In this way, the proportion between a_1 and a_2 changes with increasing intensity (Fig. 7). With the weakest stimulus, the amplitude of a_2 tends to be greater than that of a_1, but the proportion is reversed with the strongest stimulus. Although the values vary considerably from subject to subject, the tendency is the same in all cases. At the lowest and highest intensities the differences between the amplitudes of a_1 and a_2 were found to be significant at the level of $P=0.05$ (Fig. 7).

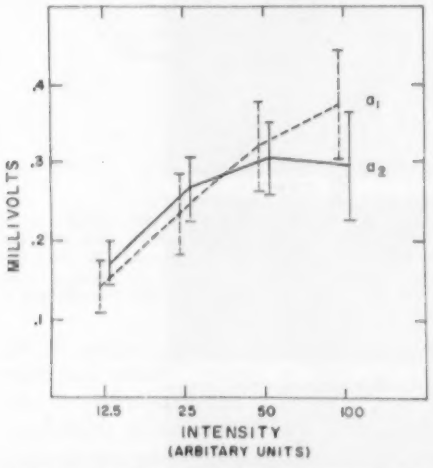

Fig. 6.—Influence of stimulus intensity on the amplitudes of the negative waves a_1 and a_2. The curve and the vertical bars (I) represent mean values and standard deviations of five normal controls. Interrupted line, a_1. Solid line, a_2. Light source was a gas-discharge lamp.

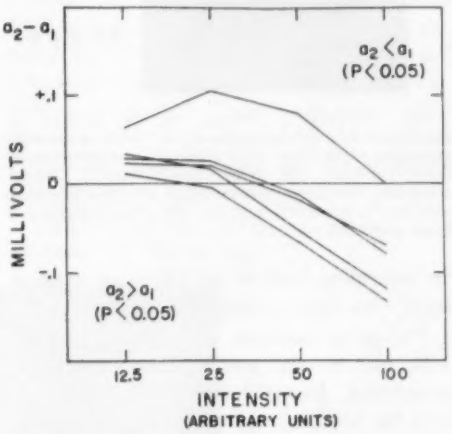

Fig. 7.—Influence of stimulus intensity on the difference between the amplitudes of a_1 and a_2. With the weakest stimulus, the amplitude of a_2 exceeds a_1, but the proportion tends to be reversed with stronger stimuli. The curves represent the individual values of five normal controls. Light source was a gas-discharge lamp.

Comment

The pattern of the human ERG elicited by strong brief flashes, and recorded with high

speed, was described in some detail by Cobb and Morton.[5,6] In the present study similar records were obtained, and the values for the time relationships of the initial response are in agreement with the figures given by these investigators.

The latency of the a-wave with the highest stimulus intensity used in this study was found to be as small as 2.6-3.0 milliseconds. Though the latency appeared to lengthen with decreasing stimulus intensities, this finding must be evaluated cautiously. Since latency reflects the time required for the a-wave to become of measurable size, the decrease in amplitude with lower intensities makes latency measurements difficult, with the error in the direction of longer latencies. Implicit time measurements, however, are more accurately determined; in the case of a_1, a_2, and the positive humps the implicit time was definitely lengthened with decreasing stimulus intensities.

It seems of interest that there is much less intersubject variability for implicit times than for amplitudes. Variations of amplitude are dependent in part on individual differences in the skull and tissue structures and their effectiveness as volume conductors.[7] The temporal course of the ERG, not being influenced by these factors, actually displays less variability.

A major problem in the study of the a-wave is the interpretation of the "break" which splits it into two parts—a_1 and a_2. Is this a "step" produced by a second negative wave of longer latency or a genuine positive hump interfering with the a-wave? On the basis of experiments with stimuli of different spectral composition, and with changing conditions of adaptation, Armington, Johnson, and Riggs[1] inclined toward the first possibility. They postulated a photopic a-wave (a_1) with a short latency, followed by a scotopic a-wave (a_2) of longer latency. Nevertheless, these authors expressly left open the possibility that the "broken" appearance of the a-wave may be caused by a genuine positive component. Their observation that the "break" tends to resemble a positive peak with shorter stimuli (about 5 milliseconds) is confirmed in the present study with ultrashort light flashes (about 10 microseconds).

A previous investigation[8] demonstrated that the a-wave in this color-blind subject represented a pure scotopic component, with complete absence of photopic activity in the negative and positive waves. The theory which suggests that there are two a-waves differing in latency (photopic, a_1; scotopic, a_2) assumes that the a-wave latency of the totally color-blind subject is in the order of the implicit time of the "break" in the normal subject (i. e., 17-20 milliseconds). In fact, it was found to be identical with the latency of the a_1-wave of the normal subject (3 milliseconds). This indicates that the photopic and scotopic components of the a-wave are not distinguished by different latencies.

The findings in the totally color-blind subject do not rule out the existence of a photopic and a scotopic component in the a-wave of normal subjects nor the possibility that such components may differ in their temporal course. The existence of a photopic and scotopic component in the negative a-wave has been proved by several investigators in dark-adaptation studies with white and colored stimuli; the demonstration of equal latencies for these two components merely argues against a theoretical explanation of this fact in terms of different latencies. However, it is possible that the latency of both components may be the same and yet the photopic process predominates in the initial part of the a-wave and the scotopic component predominates in the later part of the a-wave.† The "break" might result from the interference between two such negative processes or be due to a true positive deflection. Another possibility is that there are really two a-waves of differ-

† Unfortunately, the temporal course of the photopic component of the a-wave cannot be derived from the ERG of the night-blind subject, because in this particular subject the a-wave consists of both photopic and scotopic components.[8]

ent latencies but that each represents both photopic and scotopic activity.

A critical survey of results in past and present a-wave studies suggests that the phenomenon of the double-peaked a-wave is most likely caused by a positive hump. The behavior of the "break" with different stimulus intensities, and its temporal relations to the later positive humps, could best be explained if the "break" represented one of a series of positive humps (h_1, h_2, and h_3). This interpretation would still be consistent with any theoretical difference in the temporal course of the photopic and scotopic a-wave processes. On the other hand, it would explain the fact that in animals the presence or absence of double a-waves seems to have no correlation with functional or structural duality. The douroucouli, for instance, is a nocturnal animal with pure scotopic function which is reported to have a split a-wave.[9]

The occurrence of multiple "small waves," "wavelets," or "humps" superimposed on the positive b-wave can be seen in the ERG of several species, but their origin may differ. In the frog such multiple wavelets occur occasionally and have been attributed, in part, to the effect of synchronized impulses.[10] The effect is more marked in the cone-dominated retina of the turtle [11,12] and the pigeon.[11,13] As far as the mammalian retina is concerned, the phenomenon can be seen in the human, as first described by Cobb and Morton,[6] and in the cat.[14] A similar phenomenon in the rabbit is related to the position of the electrodes on the eye and has been ascribed to ganglion-cell activity.[15] In this connection, it is of interest that oscillatory potentials picked up from the frog's eye by intraretinal microelectrodes seem to be produced by tangentially orientated structures of the inner nuclear layer.[16]

The present investigation demonstrated a striking regularity in the temporal relationships of the periodic humps in the ascending limb of the positive ERG response of different subjects. They may represent the positive phases of a regular oscillatory response (frequency about 140/second). The possibility that the first hump occurs during the a-wave, and therefore is responsible for the split appearance of this wave, should stimulate further studies on the phenomenon of the "small waves."

Summary

The electroretinogram elicited by brief high-intensity flashes was recorded with high speed in five normal controls, in a totally color-blind subject, and in a congenitally night-blind subject.

The initial electroretinographic response in normal controls showed two negative waves (a_1 and a_2) followed by several positive humps in the ascending limb of the major positive response. With increasing stimulus intensity there was a decrease in implicit time of the negative waves and positive peaks. The latency of the initial negative response was similar in normal controls and in subjects with total color blindness and congenital night blindness.

The implications of these findings are discussed. Previous analyses of the photopic and scotopic components of the a-wave in terms of different latencies appear to be inadequate, but other differences in the temporal course of the two components cannot be excluded. The results suggest that the "break" splitting the a-wave into two portions (a_1 and a_2) may be due to a genuine positive hump leading a series of later humps, which represent a type of oscillatory response.

Ophthalmology Branch, National Institute of Neurological Diseases and Blindness (14).

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News and Comment

ANNOUNCEMENTS

Society of Military Ophthalmologists Meeting.—The Society of Military Ophthalmologists and the Society of Military Otolaryngologists will hold their annual joint dinner meeting during the meeting of the American Academy of Ophthalmology and Otolaryngology in Chicago in October.

Cocktails and dinner will be served in a private dining room of the Palmer House Hotel at 6:30 p. m. on Tuesday, Oct. 15, 1957. All members, ophthalmologists and otolaryngologists on active duty, or retired, are invited to attend.

Reservations may be made at the time of registration for the Academy Convention. Other information may be obtained from Major Stanley H. Bear, 3810th U. S. A. F. Hospital, Maxwell Air Force Base, Ala.

Postgraduate Course in Ophthalmic Surgery.—An intensive postgraduate course in Ophthalmic Surgery will be given by the Staff of the New York Eye and Ear Infirmary, covering retinal detachment surgery, cataract surgery, plastic surgery, motility surgery, glaucoma surgery, etc. There will be didactic lectures as well as demonstrations in the operating room. The course will be held Nov. 18 through 25, 1957; the fee is $250.00. For details, contact Mrs. Mabel Stewart, 218 2d Ave., New York 3.

Familial Persistent Pupillary Membranes

JOHN R. CASSADY, M.D., South Bend, Ind., and ARTHUR LIGHT, M.D., Chicago

Persistent pupillary membrane remnants are so common that they are seen frequently by every ophthalmologist. They are usually of no clinical significance. However, rare cases may show extensive membrane remnants with associated lens opacities, producing visual disturbances.

The purpose of this paper is to describe a patient with an almost complete persistence of the pupillary membranes bilaterally, progressive lens opacities, and increased corneal diameters. The relatives and ancestors of this patient had a high incidence of persistent pupillary membranes and associated anomalies. The fact that this type of iris anomaly can have an hereditary basis is not generally recognized.

The incidence of persistent pupillary membrane has been given by Stahli [1] as 30.6%. In addition to the classical strands arising from the collarette, minute remnants on the anterior capsule are extremely common (Rumbaur),[2] being seen in 35% of cases. There is a wide variety of manifestations of this type of iris anomaly, which have been classified by Ida Mann [3] as follows:

1. Complete persistence of the pupillary membrane as it existed at the maximal fetal development, the vessels containing circulating blood. This is very rare.

2. Complete persistence, but without blood in the vessels. This is more common, but also very rare.

3. Incomplete persistence. This is very common and is seen in several forms.

(*a*) A few fine strands arising from the collarette without adherence to the lens capsule.

(*b*) A persistence of capsulopupillary vessels extending from the lesser circle of the iris to pass through the pupil and behind the lens.

(*c*) Strands from the collarette adhering to the anterior surface of the lens; often there is an associated anterior capsular or pyramidal cataract.

(*d*) Strands on the anterior surface of the lens, with or without cataract, but not involving the iris.

(*e*) Projection of the iris stroma beyond the pigment epithelium at the pupil.

(*f*) Opacities of the lens capsule without strands. These appear as stellate pigmented cells.

The family to be described demonstrates several varieties of pupillary membrane remnants which coincide with this classification.

Ida Mann stated that there is very little evidence of an hereditary factor determining the persistence of pupillary membrane remnants. This opinion agrees with Brückner's [4] earlier denial of the occurrence of familial pupillary membranes.

A review of the literature reveals a few reports of familial pupillary membrane, which would seem to suggest that a genetic basis exists at times. In 1925 Pagenstecher [5] reported a family with various forms of persistent pupillary membranes in 10 of 14 members, extending over three generations. There were associated abnormalities in some of these cases. Seven of the ten with iris abnormalities had some form of congenital cataract, and two of the cases had increased corneal diameters without associated elevation of intraocular tension. Pagenstecher did not consider his cases to be cases of persistence of the pupillary membranes but preferred to call the condition a partial doubling of the mesodermal part of the iris. The outstanding feature of his cases was a continuation of mesodermal tissue from the collarette to overlie and extend beyond the pupillary margin to insert on the anterior lens capsule in a circular pattern, with or without associated lens opacities. In 1951 Wagner [8] reported a patient who was a

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member of the fourth generation in the family previously described by Pagenstecher. This patient had the same strands of mesoderm from the collarette to form a circle on the anterior lens capsule. There were, in addition, progressive lens opacities bilaterally. Both the lens opacities and the iris abnormalities appeared in an identical manner in the patient who is to be described in this paper. However, our patient had, in addition, abnormally large corneal diameters. Duke-Elder [6] and Vogt [7] classified an identical-appearing iris anomaly as a hyperplasia of the anterior layer of the iris stroma, suggesting it to be an actual hyperplasia of pupillary membrane remnants. Waardenburg [9] wrote of a woman and her two children all of whom had extensive persistent pupillary membrane remnants. Berliner [10] mentions persistent pupillary membrane in four members of a family of eight.

Our patient and her family show a marked similarity to the family reported by Pagenstecher [5] and Wagoner.[8] Therefore, it is hoped that a correlation of these two families will serve as further evidence that persistent pupillary membrane remnants can be hereditarily determined.

Report of a Case

The patient was a 3-year-old white girl (D_6), who was first seen Oct. 26, 1955, with the referring diagnosis of left congenital cataract and possible bilateral congenital glaucoma. There was a history of a slight left esotropia beginning at 18 months of age. The patient had been noticed to avoid bright lights for the six months prior to our examination. On admission, there was 10 degrees of left esotropia. The corneas were clear, 13 mm. in diameter, and without breaks in Descemet's membranes. Both anterior chambers were deep. The iris in each eye was light blue, with many light gray filaments extending from the collarette to insert in a circle on the anterior lens capsule (Figs. 1 and 2). The pupils reacted to light, apparently independently of the overlying strands from the collarette to the lens. However, the mesodermal strands appeared to interfere with complete mydriasis.

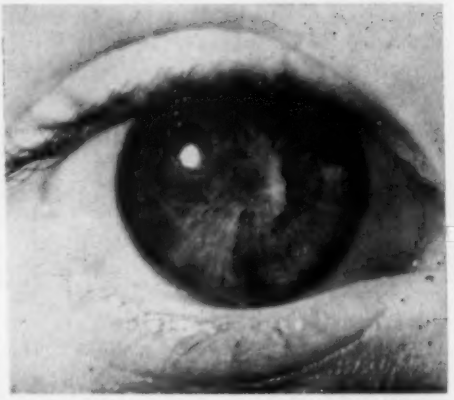
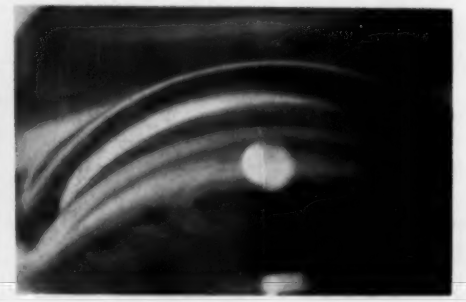


Fig. 2 (Case D_6).—Appearance of the right eye preoperatively, showing a large cornea, extensive pupillary membrane remnants, and anterior subcapsular lens opacity.

Fig. 1 (Case D_6).—Appearance of the left eye preoperatively, showing a large cornea, extensive pupillary membrane remnants, and anterior subcapsular lens opacity.

Fig. 3 (Case D_6).—Goniophotograph of the left eye, showing mesodermal strands extending from the iris surface to form a membrane over the trabecular area.



Gonioscopy revealed a translucent membrane extending from the base of each iris to Schwalbe's line, covering the trabecular area (Fig. 3). Through the membrane, blood could be seen in the canal of Schlemm, which appeared to be rather far anterior in the trabecular area. The tissue covering the trabecular area appeared to be a direct continuation of the heavy-looking mesodermal strands extending both centrally and peripherally from the iris collarette. Tension measured 15 mm. in the right eye and 11 mm. in the left eye (Schiøtz). The right lens was clear, and the fundus was normal. The left lens appeared opaque, with roughening of the anterior capsule,

Fig. 4 (Case D_8).—Postoperative appearance of the left eye, showing a white crescent of anterior capsule remaining, with persistence of the attachment to the pupillary membrane inferotemporally and inferonasally.

and no red reflex could be obtained. The patient was discharged without treatment, with a plan to reexamine her in six months. It was felt that she had had glaucoma at one time but that the tension had become spontaneously compensated in her further development.

On April 17, 1956, the patient was examined again. There was no change from the initial examination, except that the right lens had developed an anterior capsular opacification similar to that seen previously in the left eye and which obscured any fundus detail. Because vision in both eyes was now reduced, a linear extraction combined with a full iridectomy and radical iridotomy was performed on the left eye on July 20, 1956. The anterior lens capsule in the pupillary area was so thick and hard that it had to be cut with Katzin's corneal scissors. A circle of anterior capsule was cut out centrally, revealing some opaque cortical material underlying the pupillary area, with clear cortex in the periphery of the lens. On manipulation of the iris, the pupillary membrane was seen to fill partially with blood. A radial iridotomy was done inferiorly to assure a wide pupillary opening. The postoperative course was complicated by a moderate hyphema, which absorbed within two days. One month

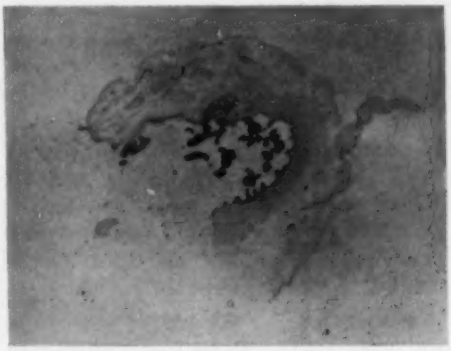
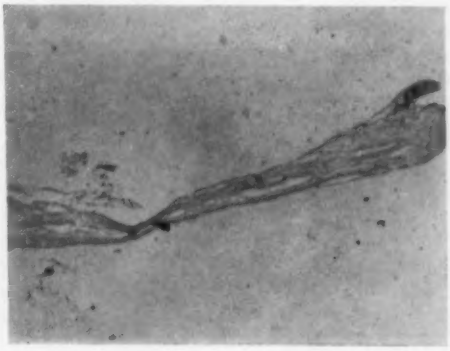


Fig. 5 (Case D_8).—Section of iris, showing a thin heavily nucleated process extending from the anterior surface of the iris at the level of the iris sphincter.

Fig. 6 (Case D_8).—Section of lens capsule, showing an intact capsule with absence of the lens epithelium. The epithelium is replaced by a layer of dense fibrous tissue. At one end of the section there is a small portion of vascular pupillary membrane remnant adherent to the capsule.



postoperatively the eye was pale, with a clear cornea and deep anterior chamber; there were no aqueous rays or cells. A large opening in the anterior lens capsule revealed an intact clear posterior capsule (Fig. 4). Inferiorly, two heavy bands of tissue extended from the collarette to the anterior capsule remnants. The fundus could be seen with good clarity, revealing an Elschnig Type I disc, with normal retinal vessels and macula. Refraction was +9.00+1.00× 90 degrees.

The microscopic examination of the iris fragment and portion of the lens capsule removed from the left eye was interesting (Figs. 5 and 6). The iris was very vascular, with many thin-walled vessels in the stroma. These vessels consisted of endothelial tubes, resembling the pattern of embryonal vessels. The small tag of membrane protruding from the surface of the iris showed a compact stroma with many nuclei. The lens capsule appeared to be intact and normal. Beneath this, the lens epithelium was replaced by a layer of avascular, partially hyalinized, connective tissue. A small remnant of pupillary membrane was adherent to the lens capsule. This consisted of an endothelial-lined tube containing a few red blood cells with very little connective tissue support.

Survey of Patient's Relatives

Several relatives of our patient had congenital cataracts, and others had glaucoma. Both the patient's paternal aunt and her greatuncle were former patients at the Illinois Eye and Ear Infirmary. In reviewing their charts, we found that an abnormality of the iris collarette had been described in both patients. With this suggestion of an hereditary factor, 18 members of the family, including our patient, covering four generations, were examined. Ten had iris anomalies; three had congenital cataracts; one had glaucoma, and three had increased corneal diameters (Fig. 7).

The first generation consisted of one person (A_1), the paternal great-grandfather of our patient. He was 75 years old, and he had been operated on for bilateral senile cataracts in March, 1945. His hospital record noted "hyalinization of the sphincter"

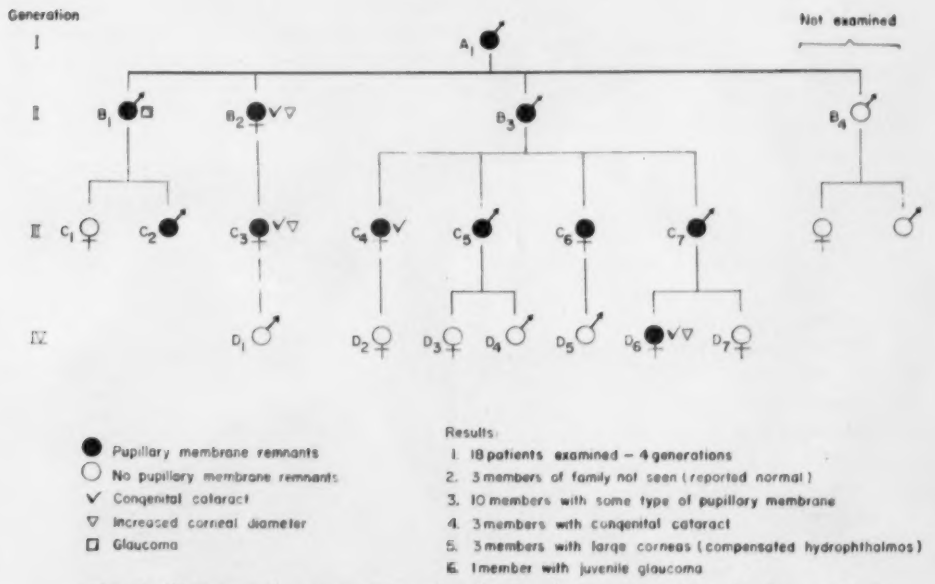


Fig. 7.—Diagrammatic representation of a family of four generations showing persistent pupillary membranes, enlarged corneas, and congenital cataracts.

Fig. 8 (Case A_1).—Aphakic right eye, showing a heavy iris collarette inferior to the pupil with a small pupillary membrane remnant inferonasally.

Fig. 9 (Case B_1).—Right eye, showing single pillar iridencleisis, with a very heavy iris collarette and a small pupillary membrane remnant at 2 o'clock.

of each iris. On July 1, 1956, we found a visual acuity of 20/40 in the right eye. A heavy iris collarette could be seen around the inferior half of the pupil (Fig. 8). The left eye had a visual acuity of 20/200, owing to moderately dense residual capsule.

The second generation consisted of the four children of A_1. Three of these children were included in our study. B_1 was a 41-year-old man who had been blind from glaucoma since the age of 27. He had been a patient at the Infirmary in 1943. He was admitted with a history of intermittent severe headaches for three months and a sudden loss of vision just prior to admission. Initial examination revealed a visual acuity in the right eye of 2/200 and light perception in the left eye. Ocular tension was 50/62 (Schiøtz). Both corneas were clear and of normal size. The iris in each eye was described as having "proliferation of tissue at the collarette." Gonioscopy showed "mesodermal tissue filling the angles bilaterally." Advanced glaucomatous cupping was present bilaterally. On Feb. 19, 1943, an iridencleisis on the right eye was performed and tension became normalized. On July 1, 1956, we examined this patient and found vision in the right eye limited to light perception with poor projection. The cornea was clear, a small functioning Type II iridencleisis bleb was present, and tension was soft. The iris had a heavy collarette, with a fine strand of tissue extending into the anterior chamber from it at about 2 o'clock (Fig. 9). An immature senile cataract was present, with a few small triangular pigmented cells on the anterior capsule in the pupillary area. The disc showed advanced glaucomatous cupping and atrophy.

Fig. 10 (Case B_1).—Left eye, showing a very heavy iris collarette, with heavy mesodermal strands on the anterior iris surface.

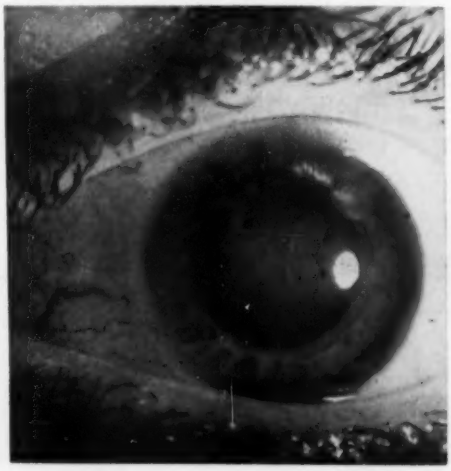


Fig. 11 (Case B_6).—Right eye, showing a thick elevated collarette, with fine pupillary membrane strands spanning the pupil.

Fig. 12 (Case B_4).—Left eye, showing a thick elevated collarette, with fine pupillary membrane strands spanning the pupil.

The left eye had no light perception; the corneal epithelium was edematous and tension was high. The anterior chamber was very deep, and the iris collarette had an extremely heavy appearance, resembling that of the right eye (Fig. 10). Gonioscopy revealed many iris processes but not the distinct mesodermal membrane seen in a classical case of congenital glaucoma. Dense nuclear sclerosis of the lens obscured any view of the fundus.

B_2 was a 44-year-old woman with history of having had surgery for bilateral congenital cataracts at 4 years of age, with resulting functional vision until she was 17 years old, when she became blind. Her right eye had been enucleated in 1949, after she had fallen and ruptured the globe. Examination on July 1, 1956, revealed vision of light perception. The cornea was involved by severe bullous keratopathy and had a diameter of 16 mm. No iris detail could be made out, owing to the corneal opacity. The ocular tension was normal.

B_3 was a 52-year-old man with a vision of 20/100 in each eye caused by immature senile cataracts. Corneal diameters and ocular tensions were normal. Each iris showed a very heavy collarette, with fine strands of pupillary membranes spanning the pupils (Figs. 11 and 12). The membranes did not touch the lens capsule. However, several characteristic triangular pigment deposits were present on the anterior lens capsule.

The third generation consisted of two children of B_1, one child of B_2, and four children of B_3. C_1, a 13-year-old daughter of B_1, had no eye pathology and no evidence of pupillary membrane or a heavy collarette. C_2 was an 11-year-old son of B_1 who had normal corneal diameters and normal intra-

Fig. 13 (Case C_3).—Right eye, showing a marked prominence of the collarette.

Fig. 14 (Case C_3).—Left eye, showing the characteristic heavy collarette, with a few fine pupillary membrane remnants arising from the temporal edge of the collarette.

ocular tensions. A heavy iris collarette was present in each eye, with a few floating pupillary membrane strands attached to the temporal collarette in the left eye and triangular pigment deposits on the anterior surface of the lens in the right eye (Figs. 13 and 14).

C_3, a 19-year-old girl, was the only child of B_2. Her left eye was enucleated at the age of 4 years because of possible tumor. In October, 1945, at the age of 8, she became a patient at the Infirmary, being referred with a diagnosis of congenital cataract and glaucoma. The corneal diameter was 15 mm., without breaks in Descemet's membrane; a mature cortical cataract was present, and "the iris sphincter was abnormally well-developed." Preoperative tension measured only 28 mm. Hg, but an iridencleisis was recommended and performed, followed one week later by a linear cataract extraction. On Sept. 20, 1956, she was reexamined and found to have a visual acuity of 20/100. The cornea was clear and 15 mm. in diameter. There was a small nonfunctioning, Type III, iridencleisis bleb at the inferior limbus. The tactile tension was normal. The lens showed a clear intact posterior capsule, with a Soemmering ring formation. The iris collarette was difficult to distinguish because of an inversion of the pupillary margin (Fig. 15). From one area a strand of tissue could be seen extending from the iris surface to the anterior lens capsule inferiorly. The fundus was seen with good clarity, revealing an Elschnig Type I disc and no fundus pathology.

The four children of B_3 all showed abnormalities of the iris. In addition, one daughter (C_4) had congenital cataracts. She had a history of good vision until the age of 13, at which time vision in the left eye became worse within a period of one year. When she was first seen at the Infirmary, at the age of 14, in September, 1951, vision in the left eye was reduced to hand movements; the cornea was not enlarged; tension was low; the iris showed a "rough pupillary border," and the lens presented an almost mature cortical cataract with an irregular surface of the anterior capsule. In April, 1952, a linear cataract extraction was attempted, but an intracapsular extraction resulted, with loss of vitreous. The operative report stated that the anterior capsule could not be cut or torn with forceps or a cystitome because of its extreme toughness. Our examination of the left eye in September, 1956, showed vision limited to light perception and good projection; corneal diameter was normal; tension was normal, and the pupil was completely updrawn, with part of a heavy collarette just visible below the

Fig. 15 (Case C_3).—Right eye, showing a small firm iridencleisis bleb inferiorly, with a thin pupillary membrane remnant inserting on the lens capsule remnants inferotemporally.

Fig. 16 (Case C_4).—Left eye, showing a markedly updrawn pupil, with a heavy collarette around the inferior pupillary margin.

Fig. 17 (Case C_4).—Right eye, showing an iridectomy superiorly, with a heavy iris collarette around the pupillary margin. There are a few fine pupillary membrane remnants inferiorly arising from the collarette; nasally a heavy white plaque of fibrous anterior capsule can be seen.

upper limbus (Fig. 16). The right eye of C_4 when she was first seen at the Infirmary, in September, 1951, had a vision of 20/70; the cornea was not enlarged; tension was 14; the iris showed a "rough pupillary border" similar to the other eye, and the lens contained club-shaped blue and punctate opacities in all layers of the periphery. During the next 13 months the cataract progressed to an almost completely mature cortical cataract. At this time, a linear cataract extraction was performed, with use of scissors to cut the hard, thick, and irregular anterior capsule, which was similar to that previously experienced in the left eye. Our examination in September, 1956, showed corrected vision of 20/30 in the right eye; normal corneal diameter; normal tension; Type I disc; a heavy collarette with a fine pupillary membrane extending from it and complete iridectomy superiorly,

Fig. 18 (Case C_3).—Left eye, showing a very heavy-appearing iris, with many iris crypts and a prominent collarette. The right eye had the same appearance.

Fig. 19 (Case C_4).—Right eye, showing the same type of heavy collarette, with a fine web-shaped pupillary membrane spanning the pupil.

Fig. 20 (Case C_6).—Left eye, showing a heavy collarette, with a fine pupillary membrane extending from the collarette superiorly.

Fig. 21 (Case C_7).—Right eye presenting a heavy collarette, with a single strand of persistent pupillary membrane spanning the pupil.

and clear posterior lens capsule with white remnants of the anterior capsule encircling the central openings (Fig. 17).

C_5, a 20-year-old son of B_3, was examined July 1, 1956. He had a very thick, heavy-looking iris with a prominent collarette (Fig. 18). There were a few fine triangular pigment deposits on the anterior capsule of both lenses. C_6, a 23-year-old daughter of B_3, was examined July 1, 1956. She displayed a heavy iris collarette bilaterally, with a fine web-shaped pupillary membrane spanning the pupil in each eye (Figs. 19 and 20). There were triangular pigment deposits on the anterior lens capsule of the left eye. There were a few fine club-shaped cortical opacities in the periphery of each lens. The corneal diameters and the ocular tensions were normal. C_7, a 25-year-old son of B_3, was the father of our patient. He had a heavy iris collarette bilaterally, with a single strand of membrane spanning the pupil without contact with the lens (Fig. 21). There were a few strands extending free from the collarette in the left eye. The corneal diameters, ocular tensions, and lens and fundus examinations were all normal.

The fourth generation consisted of seven members. Of these, only our patient (D_6) showed any abnormality of the iris, lens, or corneal diameters.

Comment

The histologic pattern of the iris reported in our case was identical to that reported by von Szily.[11] Clinically, in his case, heavy strands extended centrally from the collarette to form a circle beyond the underlying pupillary margin but remained free from the lens. Vogt[7] classified the case of von Szily[11] as a hypertrophy of the mesodermal tissue of the iris, stating that the collarette of the iris is always formed by physiologic hypertrophy of tissue rests of the pupillary membrane. Duke-Elder[6] agreed with this and added that the tissue is vascular and resembles iris stroma except for being more densely nucleated and thicker.

Wäzold[12] suggested that lens opacities at the site of contact of pupillary membranes are probably due to local metabolic disturbances in the lens. However, in view of the late onset of the cataracts in his patient and her mother, Wagner[8] felt that the lens opacity is more likely due to prolonged mechanical traction on the lens surface by the membrane, resulting from repeated pupillary movements. Our patient, D_6, had rapidly progressive lens opacities and ex-

tensive capsular and subcapsular fibrosis, suggesting that Wagner's[8] theory of mechanical traction on the capsule might have been applicable in this case. If metabolic disturbance in the lens, resulting from the attachment of the membranes, were the cause of the lens opacities in these two cases, one would expect the cataractous changes to have existed at birth or to have appeared very shortly thereafter. Because the lens opacities were of late onset, it would seem likely that the repeated traction on the lens capsule could finally result in the fibrous metaplasia of the lens epithelium and the progressive development of cataractous changes. The operative report of C_4, our patient's aunt, described a very hard, thick lens capsule, which would suggest that a similar process had taken place in her lens. However, there was no report of connection between the iris and the lens capsule prior to surgery in this case, and no connections were noticed at our examination. Also, the early cataractous changes in the right eye were cortical rather than capsular. Therefore, traction in the lens capsule can hardly be considered as the precipitating factor for the lens opacification in this case.

In comparing the family of our patient with the family reported by Pagenstecher[5] and Wagner,[8] a striking similarity is noted. In this family, 11 of 15 members showed pupillary membranes; 7 had congenital cataracts, and 2 had enlarged corneas. Of our family, of 18 members, 10 had pupillary membranes; 3 had cataracts; 3 had large corneas, and 1 (B_1) had juvenile glaucoma. The Pagenstecher-Wagner family had iris anomalies which were much more pronounced than those in our family except for D_6. The relatives of D_6 showed only small remnants of pupillary membranes, which would not be considered unusual in themselves if it were not for the hereditary pattern. In these two families, the persistence of pupillary membrane remnants in varying degrees seems genetically determined.

There were five members in the two families with abnormally large corneas but without elevated ocular tension at the time of examination or evidence of glaucomatous optic atrophy. Therefore the question arises whether these were cases of megalocornea or whether they had had increased ocular tension for at least a period in their development. Certain findings in our patient (D_6) with enlarged corneas and normal tension would suggest that she might have had a true case of congenital glaucoma which had become spontaneously compensated in some way. The gonioscopic picture was identical with that seen in many cases of established congenital glaucoma. Also, at surgery the corneoscleral tissue was found to be extremely thin at the limbus, suggesting that the tissue might have been stretched by increased intraocular pressure at some period during development. Only one member of our family (B_1) was ever proved to have glaucoma. His corneal diameters were not increased, and he was aware of no difficulty until he was 27 years old. Gonioscopy revealed many iris processes, but not the mesodermal membrane covering the trabeculum which is seen in congenital glaucoma. Thus the patient might be considered a case of juvenile glaucoma in which the iris processes were incidental. There was only one case of glaucoma in the Pagenstecher-Wagner family, and this was a glaucoma secondary to linear extraction of a congenital cataract.

It appears that the important hereditary feature is a persistence of the pupillary membrane in various forms. The cataracts which developed in some cases may be secondary to the contact of the pupillary membrane with the lens capsule. Associated with a failure of the pupillary membrane to atrophy and disappear, it seems likely in certain cases that the mesodermal tissue in the angle might fail to atrophy. This leaves a mesodermal membrane over the trabecular area, possibly explaining the buphthalmos.

According to Mann,[13] the pupillary membrane is at its maximum development by

the fifth month of intrauterine life. At that time there are three or four arcades of blood vessels extending beyond the ectodermal margin of the iris, with a very thin membrane of mesodermal tissue separating the vessels. These arcades are the end-bulbs from vessels lying in the developing iris stroma. At about six and a half months the most central arcades atrophy, and by seven months the second arcade of vessels begins to atrophy and the vessels become bloodless. The more peripheral arcade continues to enlarge with the central growth of the iris and finally becomes the lesser circle of the iris. By eight and one-half months the central arcades have normally shrunk, become fragmented, and disappeared. Mann [13] felt that, in cases of persistent pupillary membrane, some factor must act at about the fifth or sixth month to slow the development of the eye temporarily. This adverse stimulus retards the disappearance of the pupillary membrane, which, the optimum time having passed, does not completely disappear. Although Mann does not mention it in her speculation as to the cause of persistent pupillary membranes, a genetic factor might possibly produce a transient lag in development at this exact period, since growth rates and patterns are genetically controlled. A genetic factor must be an extremely rare cause of this growth lag; otherwise there would be more cases of familial pupillary membranes than the few cited here.

Summary

Although remnants of pupillary membranes are common, an hereditary form of pupillary membrane is not widely recognized. Eighteen members of a family covering four generations are described, showing pupillary membrane remnants as the primary abnormality in 11 cases, congenital cataracts in 4 cases, and enlarged corneas in 3 cases. One patient, showing an almost complete persistence of the pupillary membranes with rapidly progressive cataracts and large corneas is reported in detail.

The family is compared to a very similar family previously reported by Pagenstecher [5] and Wagner.[8] The same genetic trait was concluded to exist in both families.

The normal embryologic pattern of the resolution of pupillary membranes is reviewed, and possible alterations of this pattern resulting in membrane persistence are considered. Possible mechanisms for both the cataract formation and the enlarged corneas are mentioned, an attempt being made to relate both the cataracts and the large corneas to the abnormality of the iris.

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Acetylcholine Synthesis in Corneal Epithelium

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It was shown by von Brücke in 1938 [1] that corneal epithelium contains a remarkably high concentration of acetylcholine (ACh) and that corneal stroma contains almost none. The concentration in the corneal epithelium of rabbits appeared to be in the order of magnitude of 100γ to 200γ/gram and that in the stroma, of about 0.6γ/gram.

The high concentration of ACh in the epithelium is very intriguing and may be maintained by either one of the following mechanisms: ACh may originate in the perikaryon of the fifth nerve, flow down in the axon, and pile up in the nerve terminal, or it may be locally synthesized in the cornea either in the nerve terminals or in the epithelial cells. In all these events the presence of a choline acetylating system (ChAc), in the epithelium, is to be expected, although a high enzyme concentration is more suggestive of "local" synthesis of ACh and a low concentration, of axon flow. An attempt was made to obtain further information concerning this problem by determining the ChAc activity of the acetone powder of corneal epithelium.

Methods

Balfour and Hebb's [2] technique was applied. Corneal epithelium of 5 to 10 rabbits anesthetized with pentobarbital (Nembutal) and ether was removed by scraping the cornea with a Bard-Parker knife; it was at once dipped into 100% acetone at −20 C, well shaken, filtered on a cooled Buchner filter, and dried for 24 hours in a vacuum desiccator over phosphorus pentoxide. The average yield was about 13 mg. of epithelium powder from 10 eyes. The dried epithelium was weighed and extracted with cysteine-magnesium-saline * in a cooled mortar in the proportion of about 1.5 ml. saline to 20 mg. powder. The extract was centrifuged at 0 C, and the supernatant was incubated for one hour at 37 C in the presence of 2.5 mg. choline, 0.11 mg. coenzyme A (CoA), 3.1 mg. ATP, 0.006 mg. phosphate buffer, 0.02 mg. physostigmine (Eserine), 2.70 mg. KCl, 0.34 mg. $MgCl_2$, and 0.61 mg. NaF per milliliter of incubate. Acetate or citrate was added in concentrations of 5 and 30 mg. per milliliter, respectively, and the pH was adjusted at 7.2. For blanks, the whole procedure was the same except that the extract was substituted for the same volume of Mg-cysteine-saline solution. Incubation was aerobic in Thunberg tubes which were shaken 150 times per minute in a Warburg bath at 37.2 C. After incubation the mixtures were acidified, boiled for one minute, cooled, neutralized, diluted with Ringer's solution to a final volume of 12 ml., centrifuged, and decanted. The supernatant was assayed on the physostigmine-treated rectus, and ACh synthesis was determined by interpolation of standard curves obtained by adding known amounts of ACh to the blanks.

Results

The results are shown in the Table.

The high values in Experiments 6, 7, and 8 were possibly the result of utmost care in all details of the technique after low or zero values were obtained in Experiments 3, 4, and 5, apparently as a result of

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* Magnesium saline solution contained 0.9% NaCl and 0.029% $MgSO_4$. Cysteine content was 18 mg. per 5 cc. magnesium saline.

TABLE 1.—*Results*

Experiment	Eyes, No.	Powder, Mg.	Extract, Ml.	ACh Synthesis, γ/Gm./Hr.		
				Without Acetate or Citrate	With Acetate	With Citrate
1	20	26	1.5	0	173	--
2	10	14	1.2	--	206	--
3	10	17	1.2	0	0	--
4	10	17	1.2	0	0	--
5	13	21	1.2	30	32	--
6	10	15	1.2	29	456	--
7	10	12	1.2	112	1 000	248
8	20	20	1.5	--	660	--

inadequacies in the method. In Experiment 8 one tube was incubated without CoA and one without ATP. Synthesis in both cases was 27γ/gram/hour. Another tube was incubated without choline and the appropriate amount was added, after incubation, to be comparable with the blank; a synthesis of 120γ ACh/gram/hour was obtained.

Comment

The high choline acetylating activity of corneal epithelium extracts, synthesizing up to 1000γ ACh per gram of acetone-dried epithelium powder, corresponds to its high ACh content. It does not necessarily mean that the enzyme system is localized in the epithelial cell itself, as it may be present only in the nerve endings of the epithelial cells regardless of its presence or absence in the axon of the fifth nerve. In all these cases, corneal epithelium will be able to synthesize ACh, and there is no conclusive evidence in favor of any of them.

Some investigations seem to indicate a localization of the enzyme in the epithelial cell itself. Feldberg and Vogt [3] and recently Hebb [4] were unable to demonstrate the presence of ChAc in afferent fibers. The optic, the acoustic, and the posterior spinal fibers did not synthesize ACh, and the claims of de Roetth [5] concerning synthesis by the optic nerve could not be confirmed. Yet a small amount of enzyme in the axon may not be detectable but a continuous flow of small concentrations may lead to a higher concentration in the terminals, just as cutting a peripheral nerve leads to a higher concentration of ACh and ChAc in the central stump.[6,7]

An enzyme flow in the axon becomes, however, less likely in view of the experiments of von Brücke, Hellauer, and Umrath.[8] Seven days after peripheral section of the fifth nerve, the ACh content of the cornea decreased 35% to 50% and the thiamine content also diminished. Apparently the synthesis of ACh goes on after section of the fifth nerve either in the nerve terminals or in the epithelium; yet the decrease in ACh production indicates that the nerve is somehow involved in the synthesis, possibly by supplying thiamine (as suggested by experiments of von Muralt [9,10]) or ATP (as suggested by experiments of Holton and Holton [11]) or by a slow degeneration of the nerve terminals.

Although the location of the enzyme remains obscure, its presence in high concentrations in corneal epithelium makes it likely that a great deal of ACh is synthesized locally. ACh synthesis, which occurred after incubation without acetate or choline and occurred slightly after incubation without ATP or CoA, indicates that all these substances are present in corneal epithelium.

The significance of the high concentration of ACh is by no means clear, but it may be essential for the integrity of corneal epithelium. In virus, traumatic, or surgical lesions of the fifth nerve leading to an epithelial breakdown, von Brücke, Brückner, and others [12] have reported striking results on the regeneration of the epithelium by local administration of ACh. The synthesis of ACh will be impaired when the supply of any of the essential agents becomes depleted. Section or disease of the fifth nerve may block the transport of at least one of these

agents, viz., thiamine—the acetyl donor—and possibly others, e. g., ATP. Vitamin deficiencies, in particular those in which thiamine and pantothenic acid (CoA) are involved, may directly interfere with an adequate synthesis of ACh. If, therefore, ACh has an essential physiological function in the corneal epithelial cell, impaired synthesis may be the fundamental cause for epithelial breakdown in lesions of the trigeminal nerve and for the pure epithelial punctate lesions in vitamin B deficiencies.[13]

For receptors in the carotid body,[14] the tongue,[15] and the skin [15] a role in chemosensory transmission has been suggested, though the evidence for ACh as a transmitter substance cannot be considered as final.[16,17] So far, there are no indications that ACh in corneal epithelium is involved in the transmission of touch or pain stimuli. Atropine does not block and physostigmine does not enhance these sensations; neither did cholinesterase show an appreciable effect when it was instilled in high concentrations into the eyes of three rabbits and two human subjects.

Summary

Cholinacetylase has been demonstrated in acetone-dried corneal epithelium, synthesizing up to 1000γ of acetylcholine per gram per hour.

The significance of acetylcholine in corneal epithelium and the effects of its inadequate synthesis are discussed.

The ideas of Dr. A. B. L. Beznák are incorporated in this investigation.

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Paracentral Homonymous Hemianopic Scotoma

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Paracentral homonymous hemianopic scotoma is a rare field defect. Only a small number of cases have been reported. In 1938 Allen and Carmen,[1] in reviewing the literature, reported on 15 cases, including 1 of their own. Walsh,[2] in his book, devotes two short paragraphs to the subject, citing the cases reported by Allen and Carmen but adding no new cases since that time. Hughes,[3] in his book, published in 1954, has illustrations of three cases which he attributes to vascular lesions, and Harrington,[4] in his recent book, published in 1956, shows a case due to injury of the occipital lobe.

The symptom complex consists of the following elements:

1. There is difficulty in reading fixation of objects located close to the central field on the side of the defect. If the defect is to the right, one word, or only part of a word, is seen, but the following words or syllables cannot be made out. If the defect is to the left, the patient has difficulty in finding the next line.

2. Visual acuity is not impaired. The macula is always spared, and, once fixation is made, there is no difficulty in seeing the object clearly.

3. There are characteristic congruent paracentral homonymous scotomas close to, but not involving, the macula, with normal peripheral fields.

4. The optic nerves are normal, the lesion being not in the optic tracts but in the geniculocalcarine pathway or the striate cortex.

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The patient I wish to report is a 64-year-old white man who was seen by me in March, 1954. His chief complaint was inability to read.

The following past history was obtained from his physician. The patient had an arteriosclerotic cardiac condition which manifested itself in the form of a bundle branch block. The blood pressure had always been normal and the pulse rate regular, between 60 and 70.

During the year of 1952 he had had infrequent episodes of syncope which were diagnosed as petit mal and so confirmed by a complete work-up at Johns Hopkins Hospital.

His present condition had its onset on Dec. 9, 1953, when he became rather faint, weak, and dizzy. His blood pressure was normal, but his pulse rate had dropped to 28 per minute.

He was hospitalized, and on Dec. 14 the pulse rate was 24 and on Dec. 15 it was 18. At that time he developed marked visual loss—no light perception—and some mental confusion. The blood chemistry studies showed a blood urea nitrogen level of 42 mg. per 100 cc. and an NPN value of 100 mg. per 100 cc. The diagnosis was azotemia, secondary to circulatory failure in the kidneys.

Dextrose intravenously plus atropine and ephedrine were given. Two days later the blood urea nitrogen was 18 mg. and the NPN was 30 mg. per 100 cc. Vision began to clear, and on discharge from the hospital, on Jan. 1, three and one-half weeks after his admission, his only complaint in regard to vision was inability to read.

An eye examination was done on March 12, 1954. Vision in each eye with correction was 20/20. He could identify Jaeger 1

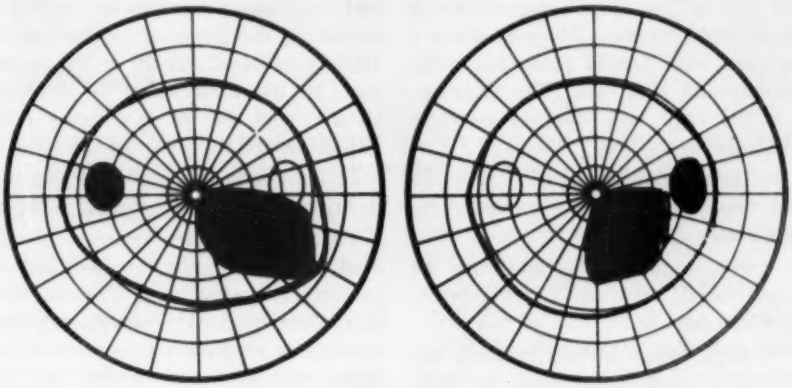

Figure 1

letters on a reading chart but had difficulty reading a word unless he spelled it out. The eye grounds were normal except for Grade I arteriosclerosis of the retinal vessels. Peripheral fields were completely normal, and central fields determined on the small screen of the Ferree-Rand perimeter showed no abnormality.

Because of the normal fields and vision, it was thought that he was suffering from some form of sensory aphasia, alexia, or word blindness and had lost the ability to recognize printed words.

He returned again 10 months later, stating that there had been no improvement, the difficulty in reading persisting. The vision was 20/20 in each eye as before, and the fundi were essentially normal. Central fields were done on a large tangent screen at 1 meter and a right inferior quadrant homonymous scotoma was found, as depicted in Figure 1. The central field shows the scotoma at 1 meter to 1 mm. white and 2 mm. red.

The patient, who was Jewish, volunteered the information that he had found no diffi-

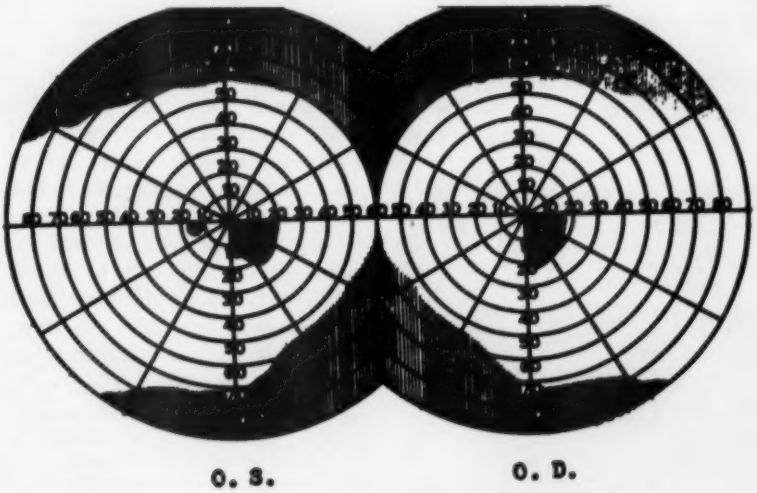

Figure 2

culty in reading Jewish newspapers, which read from right to left. He was given a reading card with Jewish print and could read rapidly and with ease. The scotomas, being to the right side, did not interfere when he read to the left.

He has been examined several times in the past two years, and the scotomas remain the same.

The lesion is uncommon and may be easily overlooked, particularly if the defect is on the left side.

The etiologic factor is undetermined but probably is a vascular accident in most cases. In my patient, suffering from a bundle branch block, the marked slowing of the pulse resulted in an impairment of kidney function, followed by retention of nitrogenous products in the blood. The complete loss of vision while he was in the hospital was undoubtedly cortical in origin, due to a uremic state. As the azotemia subsided, cortical function was restored, leaving a lesion, probably a small thrombus, resulting in the field defect.

The exact location of this lesion can only be speculated upon. It is assumed to be in the calcarine fissure of the occipital lobe or in the optic radiation. Hughes [3] states that vascular lesions producing homonymous and congruous scotomata are usually due to lesions of the posterior cerebral artery affecting the blood supply of the striate cortex. In the three cases he describes the lesions were due to thrombosis occurring in branches of the posterior calcarine artery.

In Harrington's [4] case the lesion was due to injury at the tip of the calcarine fissure.

This case has been reported in order to draw attention to the symptom complex of paracentral homonymous scotoma, which is so easily missed. The complaint of sudden inability to read, in the presence of normal fundi, normal visual acuity, and normal peripheral fields should make one suspect this syndrome.

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The Vitreous

Gross and Microscopic Observations Seen in Age and Disease, with Special Emphasis on the Role of Vitreous in Detachment of the Retina

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Introduction

There are relatively few studies of the vitreous found in the literature where emphasis is placed on the gross appearance. Many criticisms of existing studies have frozen further investigation of obvious pathologic changes seen clinically, macroscopically, and microscopically. De Wecker [1] accurately described the process of vitreous shrinkage as well as the important relationship of the vitreous to detachment of the retina. Not until Gonin [2,3] applied cautery to seal the retinal tears did the significance of vitreous changes in retinal detachments finally gain its relative importance.

The normal vitreous is a transparent gel that completely fills the posterior part of the eye. In its normal state it acts as a cushion to help keep the structures of the eye in their normal position. The important function of cushioning the retina helps maintain the retina in contact with the pigment epithelium. In the young adult in whom there has been no history of disease, the vitreous has its normal characteristics. However, with age certain changes take place in the form of shrinkage, liquefaction, and formation of strands. Such changes in themselves may have no adverse affect on the general health of the eye. On the other hand, certain relatively minor changes associated with the usual senescent developments may result in serious complications.[4-8]

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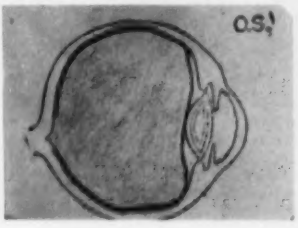
Changes in Vitreous

During the process of opening the globes sent to the Pathology Laboratory at the Institute of Ophthalmology, Presbyterian Hospital, it was observed that certain changes found in the vitreous fell into a general pattern, depending on age, history of ocular disease, or injury. These changes found in the vitreous have been previously described by many authors [9,10]; however, their relative importance has been minimized, since they have often been considered artefacts.[4-8] Further study and research led to the conclusion that most of the changes found were not artefacts but, for the most part, existed prior to enucleation.

A series of normal globes from young persons was opened, both in the fresh state and after fixation. Both the fresh and fixed globes, when opened, showed the vitreous to be transparent, normal in consistency, and at all times filling the vitreous cavity (Figs. 1, 2, 3, 4, 5, and 6).

Other globes, in which there had been a history of disease, when opened, showed typical changes in the vitreous that cor-

Fig. 1.—Diagram showing the normal relation of the vitreous to the other structures of the eye. The vitreous is lightly shaded, and the retina is a dark line. Here, the vitreous completely fills the vitreous cavity and mechanically cushions the retina against the pigment epithelium.



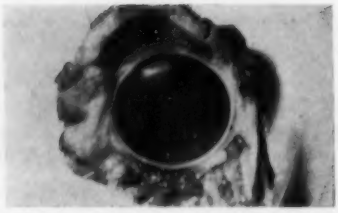
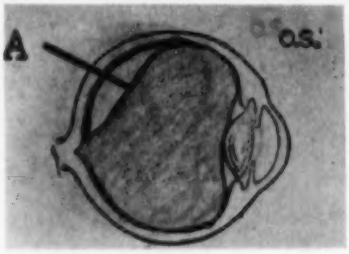


Fig. 5.—A picture of the orbital contents removed because of a malignant melanoma of the conjunctiva. The eyeball is normal. The vitreous is clear and retains its normal consistency.

responded to the type of ocular disease. Where there had been a perforating wound that extended into the vitreous cavity, it was generally possible to follow the course of perforation by a condensation and a loss of transparency of the vitreous. The course of an intraocular foreign body could often be traced by the thin line of opaque vitreous extending from the point of entry and coursing to the locale of the foreign body. In such a case, where there has been damage to the vitreous by the perforating wound or intraocular foreign body, the vitreous becomes liquefied and often leaves localized areas of condensation in the form of dense vitreous strands (Fig. 7). If there has been an associated inflammation, the strands become cellular and later contract. The contracture of the strands often results in a detachment of the retina.[17-19]

In order to interpret many of the routine changes found in the vitreous, it is necessary to study the eye both in youth and in age, because typical changes take place in the vitreous of elderly patients. As previously shown, normal young adult eyes were found

Fig. 8.—Diagram showing an early detachment and shrinkage of the vitreous (*A*). The superior detachment is the earliest change and is usually limited by the ora and optic nerve.



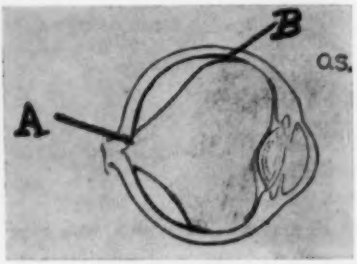
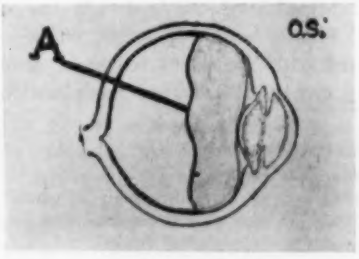


Fig. 9.—A diagram showing a somewhat more advanced shrinkage of the vitreous. However, the vitreous is still attached at the optic nerve (*A*) and near the ora (*B*).

to have normal vitreous. However, with age, there is a tendency for vitreous to shrink and become detached posteriorly.[20-26] The superior portion of the vitreous is usually the area to become detached earliest (Fig. 8). The posterior hyaloid is often visible with the slit lamp clinically. With subsequent shrinkage of the vitreous, the space created between the retina and the hyaloid is filled with a fluid substance resembling aqueous. Further detachment of the vitreous extends to the region of the equator, but the attachment of the hyaloid to the optic nerve remains (Figs. 9 and 10). The attachment of the vitreous to the optic nerve subsequently separates, and there is then a complete posterior detachment of the vitreous (Figs. 11, 12, 13, and 14).[27,28] Oftentimes, such a detachment can be seen clinically and is identified by the presence of a circular opacity undulating on the posterior surface of the hyaloid.

In the absence of previous ocular disease, such shrinkage of the vitreous may cause no

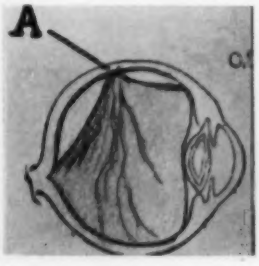
Fig. 11.—A diagram showing complete posterior detachment of the vitreous (*A*). Progressive shrinkage of the vitreous has caused detachment of the hyaloid from the optic nerve.



damaging effects to the eye. When the vitreous first becomes detached, however, there are symptomatic manifestations in the form of lightning flashes. This was first described by Moore [29] and later amplified by Verhoeff.[30] The symptoms were explained by the fact that the floating vitreous strikes against the retina following rapid movement of the eye. Such contact with the retina causes stimuli, to be interpreted as lightning flashes. Further shrinkage of the vitreous results in a disappearance of these troublesome symptoms.

Detachment of the vitreous is an extremely common finding in patients over 50 years of age. Above the age of 65 years shrinkage of the vitreous was found in between 70% and 75% of the eyes examined. Simple shrinkage of the vitreous cannot be associated with other pathological changes found in the eye, but it is apparent that some conditions in the eye may hasten or cause changes identical with those seen in the elderly person. In cases of myopia a shrinkage and liquefaction of the vitreous may develop and numerous floating strands are often seen clinically as well as macroscopically. These changes per se may cause no damage to the visual organs. However, if in the process of shrinkage there exists an adhesion of the vitreous to the retina, subsequent shrinkage of the vitreous may result in a retinal tear following traction on this localized area [32] (Fig. 15). Condensation of the vitreous with or without downgrowth of cellular elements along the strands

Fig. 15.—A diagram showing superior detachment and shrinkage of the vitreous with the presence of a retinovitreous adhesion (*A*). There is obvious traction exerted by the vitreous on the retina.



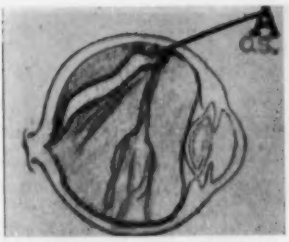


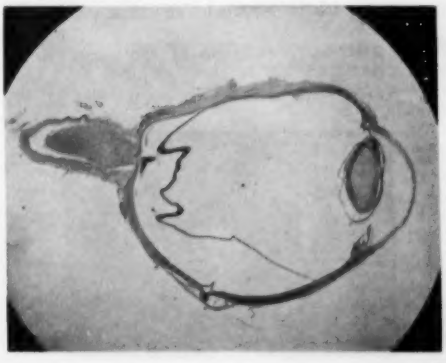
Fig. 16.—A diagram showing a situation similar to that in Figure 15 but with further shrinkage of the vitreous and a tear in the retina at the point of retinovitreous adhesion (*A*). Once the tear is present, fluid migrates behind the retina and causes a detachment of the retina. The subretinal fluid is darkly shaded.

may subsequently contract and cause sufficient traction on the retina to create a tear and later cause a detachment (Fig. 16). From the above facts, it can be concluded that the vitreous plays a dominant role in the formation of detachment of the retina.[33] Such details can be clearly seen by studying the specimen shown in Figures 17, 18, 19, and 20.

The patient is said to have developed a detachment of the retina involving the superior temporal quadrant. A horseshoe-shaped tear was found and treated with diathermy. On or about the second week, there was noted an elevation of the treated area, and, in addition, the region had a bluish-gray color. A neoplasm was suspected, and an enucleation was performed.

Examination of the globe showed the treated area of the sclera to be markedly

Fig. 19.—A photograph of the microscopic section taken through the region of the tear. The marked flattening of the sclera is obvious.



flattened (Fig. 17). When the globe was opened no tumor was found, but there was a moderately severe hemorrhage in the choroid. The retina was in place, and the area of treatment completely surrounded the tear. The vitreous was detached and shrunken but was adherent to the tear (Fig. 18).

After studying the illustrations in this case, it may be surmised that the apparent elevation was due to the fact that the sclera was shrunken and flattened, giving the ophthalmoscopic appearance of elevation (Fig. 19). The bluish-gray color was no doubt due to the choroidal hemorrhage (Fig. 20).

In spite of the ophthalmoscopic changes, the retina had been reattached and the hole had been sealed by diathermy. After treatment, the vitreous continued to remain attached to the flap of the retinal hole.

During life, small areas in the retina become atrophic. The cause of these changes is not clear, but at these points of atrophy the vitreous becomes adherent. During the process of detachment and shrinkage of the vitreous with progressive age these localized adhesions may exert traction on the thin, atrophic area of the retina, causing a tear, with subsequent detachment of the retina (Fig. 21).[34]

In addition to the localized atrophic areas in the retina, small points of chorioretinitis may be present. Such areas are associated with retinovitreous adhesions corresponding to the scar. The mild focal chorioretinitis does not produce a dense proliferative scar and therefore leaves thin weakened areas that can be easily torn by traction of the adherent vitreous (Fig. 22). These strands may be extremely dense and capable of exerting great force in the form of traction at a localized point on the retina (Figures 23A, 23B, and 24).[35]

On the other hand, the dense chorioretinal scars formed following an exudative process rarely tear following traction from the vitreous strands, because the chorioretinal adhesions are so dense that they can withstand the pull.

The vitreous strands following severe chorioretinitis may be very dense and even cellular. The contracture of these cellular strands exerts great force and may cause a separation of the retina (Fig. 25A, 25B, and 26).

Secondary Holes.—There are different types of holes found in the retina in the presence of detachment. The usual horseshoe-shaped tear is probably formed by the presence of a retinovitreous adhesion in an eye where the vitreous has become shrunken and causes localized traction on this weakened area of the retina. Once the fluid has gained access to the subretinal space, the retina separates from its contact with the pigment epithelium and becomes elevated. The fluid in the subretinal space exerts further pressure on the retina and increases the area of detachment.

If this subretinal fluid migrates from above to the lower portion of the fundus, it may cause an elevation below. Such an

Fig. 25B.—A photograph of the same eye as shown in Figure 24, but both callottes have been removed to show the dense character of the strands.

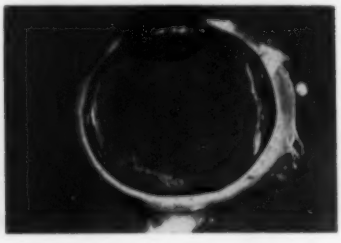


Fig. 26.—Microscopic slide of Figure 25B, which shows the cellular character of the dense vitreous strands.

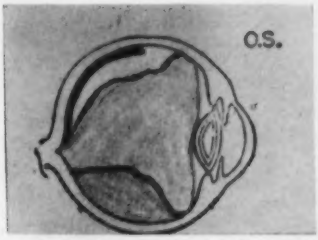


Fig. 27.—A diagram showing a hole in the retina above, with a large dependent detachment below.

elevation may be bullous, and there may be no holes in the lower portion of the retina (Fig. 27). On the other hand, one frequently sees one or more small punched-out dehiscences in the elevation. Such secondary

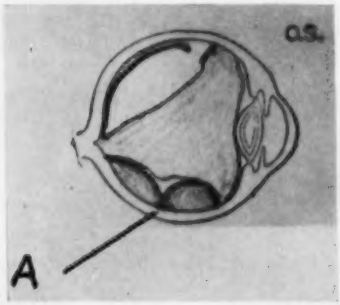
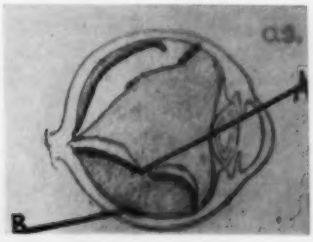


Fig. 28.—A diagram showing a hole in the retina above, with a dependent detachment below. The chorioretinal adhesion is a point where a potential secondary hole in the retina may form (*A*).

holes can be caused by the presence of small localized areas of chorioretinitis existing prior to the detachment (Fig. 28). When the fluid migrates to the lower portion of

Fig. 29.—A diagram showing a condition similar to that in Figure 28, but sufficient force has been exerted on the retina to cause a secondary hole to form (*A*) at the location of the chorioretinitis (*B*).



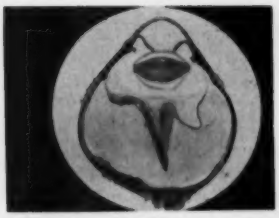


Fig. 30.—A photomicrograph of the eye of a 44-year-old woman who developed a detachment of the retina following diabetic retinopathy. The detachment was of long standing, and secondary glaucoma developed. A section of the globe shows a complete detachment of the retina.

the globe, sufficient force is exerted on the localized adhesions that either a hole is formed or the small areas of chorioretinitis are broken (Fig. 29). In this case, traction from within is not responsible for the pres-

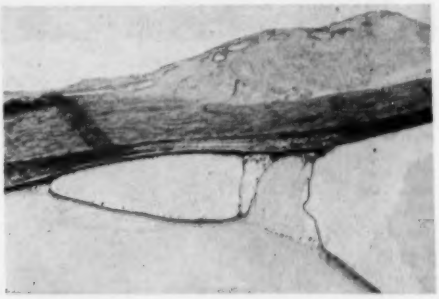
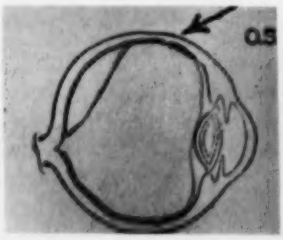


Fig. 31.—High-power photomicrograph of Area *A* in Figure 30, showing an area of chorioretinitis that has been under traction from the detached retina, causing thinning of the retinal elements. Further traction can easily cause a hole to form by a rupture of the thinned retina.

ence of the retinal holes. The force forming the secondary holes is exerted by the pushing effect of the subretinal fluid rather than by the traction from within (Figs. 30 and 31).

Fig. 32.—Diagram showing two small holes in the peripheral retina without detachment. The vitreous cushions the retina and exerts no traction on the lips of the hole.



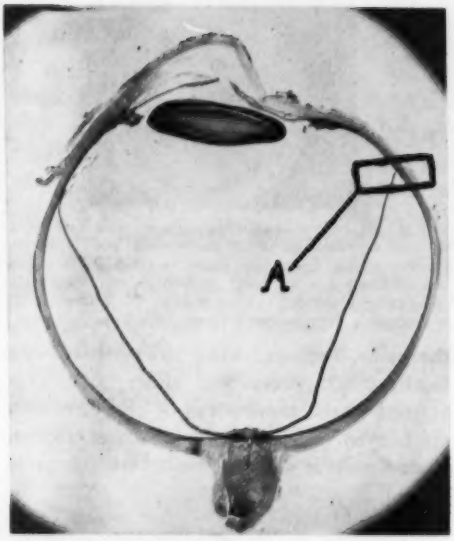


Fig. 33.—A photomicrograph of an eye taken from a 15-year-old girl who had an abscess of the cornea. There was no clinical evidence of detachment. The apparent detachment shown in the section is an artefact due to sectioning.

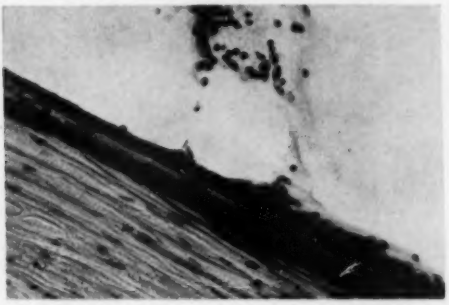


Fig. 34.—A high magnification of the peripheral retina in Area *A* from Figure 33. There is a small cyst at the ora serrata which involves all layers of the retina.

Peripheral Cysts.—In recent years, with the improved methods of examining the peripheral fundus, many small holes have been described in the absence of detachment of the retina. These holes may be the result of small cysts in the periphery. The holes may remain unchanged without ever causing a detachment so long as the vitreous cushions this area (Fig. 32). The small peripheral cysts of the retina have often been blamed for detachment of the retina. But with a better understanding of the changes found in the vitreous clinically and macroscopically, as well as microscopically, one can conclude that such cystic changes are not the major cause of detachment (Figs. 33, 34, and 35).

Fig. 2.—A picture of a freshly enucleated normal eye taken from a young person with a neoplasm of the orbit. The vitreous is crystal-clear and fills the vitreous cavity.

Fig. 3.—A picture of a normal eye removed from a middle-aged person with an orbital neoplasm. The eye and orbital contents were fixed in Gonzalez' solution. The vitreous is clear and shows no evidence of shrinkage.

Fig. 4.—A picture of a normal eye removed from a patient in his early 50's. There was no history of eye injury or disease. The vitreous is clear and fills the vitreous cavity.

Fig. 6.—A picture of a normal eyeball removed because of an orbital tumor. The expansion of the tumor has caused a compression of the globe without obvious changes in the normal transparency of the vitreous.

Fig. 7.—A picture of an eye which had sustained injury from a perforating foreign body. The course of the foreign body is made clear by the condensation of vitreous in the form of fine striae.

Fig. 10.—A picture of an eye removed after iridocyclitis and secondary glaucoma. The section shows a posterior detachment and shrinkage of vitreous, but the hyaloid is still in contact with the optic nerve.

Fig. 12.—Picture of an eye from a 65-year-old man. In childhood a blow to the eye resulted in the loss of vision. Glaucoma developed two years prior to enucleation. A section of the globe reveals complete posterior detachment of the vitreous (*A*). The hyaloid is no longer attached to the optic nerve.

Fig. 13.—Picture of an eye removed from a 60-year-old man with a history of uveitis many years previously. Secondary glaucoma developed, with subsequent loss of vision. A section of the globe shows a complete posterior detachment of the vitreous (*A*), which is somewhat more advanced than that in the previous Figure.

Fig. 14.—Picture of an eye removed from a 79-year-old woman in whom acute glaucoma developed following eye examination. Tension was not controlled, and vision was lost. A section of the globe shows advanced shrinkage of the vitreous (*A*). The cushioning effect exerted by the vitreous is present only on the peripheral part of the retina (*B*).

Fig. 17.—A photograph showing the marked flattening of the sclera above the area where treatment was applied.

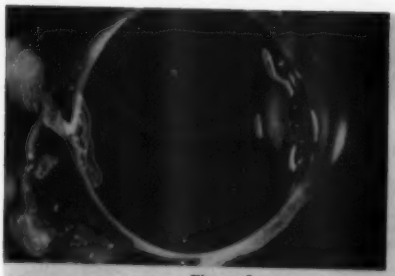


Figure 2

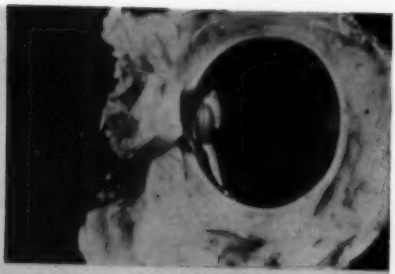


Figure 3

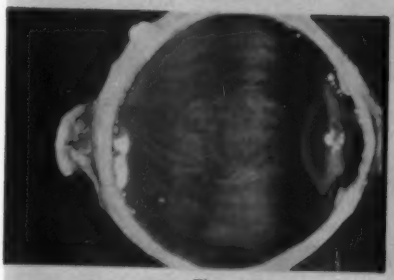


Figure 4

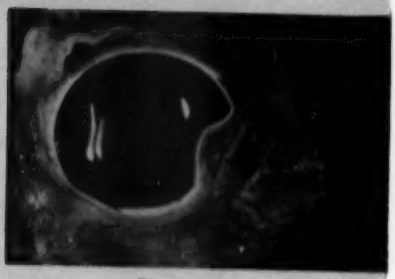


Figure 6

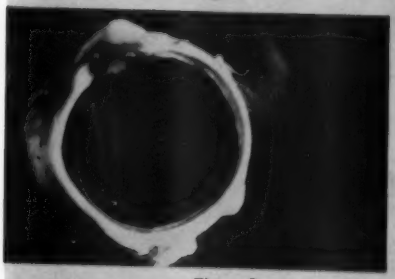


Figure 7

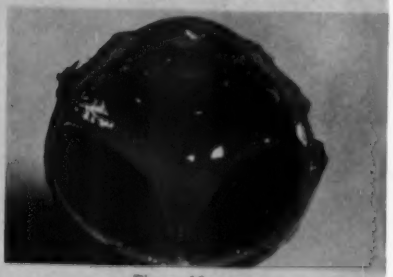


Figure 10

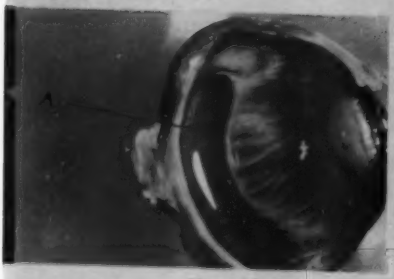


Figure 12

Figure 13

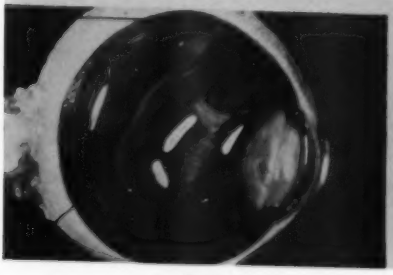


Figure 14

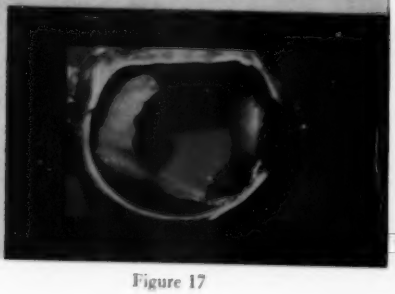


Figure 17

Fig. 35.—Serial sections of the cyst shown in the previous illustration reveal an actual hole at one point. There was an existing hole in the retina, but the vitreous cushioned this area and exerted no traction in order to cause a detachment.

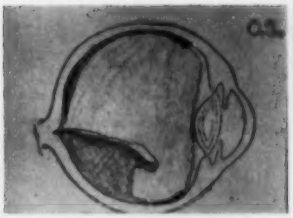


Fig. 36.—A diagram showing the vitreous protruding through a disinsertion and partially filling the subretinal space.

Disinsertion.—The mechanism of disinsertion is probably different from that of all other types of retinal tears or holes. A disinsertion frequently occurs following direct trauma to a normal eye. The retina tears from its attachment at the region of the ora serrata. As the hole is made, formed vitreous protrudes into the subretinal space through the large tear (Figs. 36 and 37). Since the tear may be plugged with vitreous, fluid does not migrate to the subretinal space. Therefore, the detachment may remain unchanged over a long period. On the other hand, the retina is separated by formed vitreous and may not respond to the usual diathermy barrage. It is usually necessary to apply diathermy applications in the area behind the vitreous in order to wall off the elevated region.

Vitreous Hemorrhage.—Associated with age and disease, vitreous hemorrhage may present itself as a part of a syndrome. In cases in which hemorrhage takes place and makes its way into normal vitreous, the blood remains dark and in dense strands, causing a prolonged decrease in vision. Ab-

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Fig. 18.—The interior of the eye, showing the shrunken vitreous adherent to the lip of the tear. The faint white spots surrounding the tear represent the diathermy barrage.

Fig. 20.—High-power photomicrograph depicting the retinal tear; beneath the retina is the choroidal hemorrhage. The tag of the retina is attached to the vitreous, as is represented by the faint-staining strands extending from the tag of the retina (*A*). Note that the retina is firmly reattached in the area of the hole.

Fig. 21.—A photomicrograph showing a localized area of retinal atrophy (*A*) with retinovitreous adhesions (*B*). Traction by the shrunken vitreous may produce a tear in the weakened retina.

Fig. 22.—A photograph of an eye taken from a 60-year-old man with history of repeated attacks of iridocyclitis associated with vitreous hemorrhage. Secondary glaucoma developed. A section of the globe shows numerous atrophic areas of the retina and focal chorioretinitis. The retinovitreous adhesions (*A*) could exert sufficient traction to tear the retina and cause detachment.

Fig. 23A.—A photograph of an eye taken from a 38-year-old man. A loss of vision developed following a blow to the eye. Glaucoma developed two years before enucleation. A section of the globe shows dense vitreous strands capable of exerting traction with great force.

Fig. 23B.—Callotte from specimen shown in Figure 23A, demonstrating scattered atrophic areas which probably represent inactive chorioretinitis. Note the vitreous adhesions to these scars.

Fig. 24.—A photograph of the eye of a 26-year-old man who developed detachment of the retina following trauma. Subsequent to operation there were repeated attacks of iridocyclitis and secondary glaucoma developed. A section of the globe shows extensive chorioretinitis with a shrinkage of the vitreous and fine vitreous strands adherent to the region of the scar.

Fig. 25A.—A photograph of an eye taken from a 47-year-old man who had repeated attacks of iridocyclitis. He later developed glaucoma. A section of the globe shows dense vitreous strands in the form of membranes which are firmly attached to the areas of chorioretinitis.

Fig. 39.—A photograph of a globe taken from a 66-year-old woman, with diabetes of long standing, who had a history of repeated vitreous hemorrhages. A section of the globe shows blood in the normal vitreous.

Fig. 43.—A photograph showing a detachment of the retina in the presence of a malignant melanoma of the choroid. Note that the subretinal fluid has coagulated following fixation of the globe.

Fig. 37.—A photomicrograph of an eye with a history of a bullous detachment following a disinsertion. The microscopic section shows the peripheral retina and disinsertion. The vitreous (*A*) extended through the disinsertion into the subretinal space when the globe was opened, but when the specimen was handled, the vitreous withdrew from the disinsertion. However, a small amount of vitreous is present in the retinal tear.

sorption of such blood may take months or even years (Figs. 38 and 39).

It is not unusual to see a patient with a recent vitreous hemorrhage that clears within 24 to 48 hours. This hemorrhage probably takes place in the retrovitreous space. Such an eye probably has a detachment of the vitreous with shrinkage, and the blood escapes into the area containing fluid behind the vitreous. Blood in this fluid medium settles rapidly, as blood in the anterior chamber quickly forms a hyphema (Figs. 40 and 41). A rapid return of vision may be experienced.

Such a mechanism is often seen prior to the formation of detachment of the retina. A small rent in the retina may tear a blood vessel and cause blood to fill the subvitreous space and obscure the findings. After the blood has absorbed or settled, one

Fig. 38.—A diagram showing hemorrhage into the normal vitreous. Blood in the gel may remain unchanged for months.

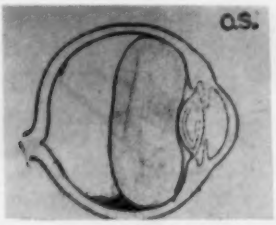


Fig. 40.—A diagram showing blood in the subvitreous space. The blood has settled into the dependent part of the globe. A gross specimen was observed, but photography was not possible, because the blood escaped with the subvitreous fluid at the time the globe was opened.

is able to find a tear in the retina and a small localized detachment.[36]

Subretinal Fluid.—Depending on the nature of the disease process, subretinal fluid has different characteristics. If there is a

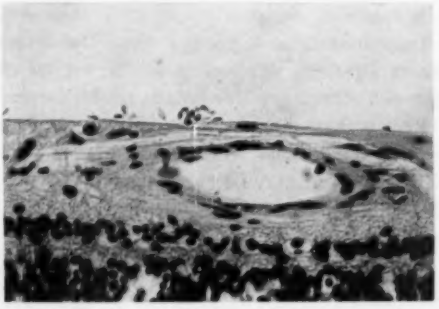
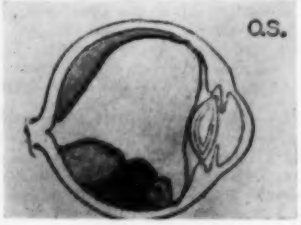


Fig. 41.—A microscopic section that shows hemorrhage on the surface of the retina following a subvitreous hemorrhage, as described in Figure 40.

serous detachment of the retina with a hole, the subretinal fluid remains liquid and does not coagulate on fixation. On the other hand, in the presence of a choroidal tumor

Fig. 42.—A diagram showing a detachment of the retina associated with a melanoma of the choroid. The subretinal fluid has coagulated on fixation.



with a secondary detachment of the retina where no hole exists the subretinal fluid is high in protein and quickly coagulates on fixation (Figs. 42 and 43). Such fluid gives a 4+ Pandy reaction, indicating an increased amount of protein. The same coagulability of the subretinal fluid is seen in inflammatory conditions causing a detachment—for example, Haroda's disease, toxemia of pregnancy, and localized chorioretinitis. Such definite differences in the subretinal fluid could very easily be a help in establishing the diagnosis in some individual instances.

Conclusion

Vitreous changes fall into definite patterns that may be confirmed by clinical, gross, and microscopic findings. Many of previously described artefacts are actually real changes in the vitreous. The vitreous plays an important part in health and disease of the eye. The mechanism of detachment of the retina is more clearly understood with our increased knowledge of the changes in the vitreous.

635 W. 165th St. (32).

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Red Glass X-Ray Spectacles

ERWIN E. GROSSMANN, M.D., and PAUL R. THORNFELDT, M.D., Milwaukee

A green-free optically ground spectacle lens has been found to be eminently successful in providing rapid dark adaptation and other conveniences to both radiologists and internists who do a great deal of darkroom studies. The problem first arose in the case of an internist specializing in heart and lung. His corrective lenses were as follows: O. D. −1.75, ax 180, and O. S. −0.50 −2.50, ax 180, add +1.75 for near. The lenses were mounted in a red plastic frame including sides (Fig. 1). Most of

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From the Department of Ophthalmology, U. S. Veterans Hospital (Wood, Wis.) and Marquette University School of Medicine.

Fig. 1.—O. D. −1.75, ax 180; O. S. −0.50 −2.50, ax 180; add +1.75 for near.

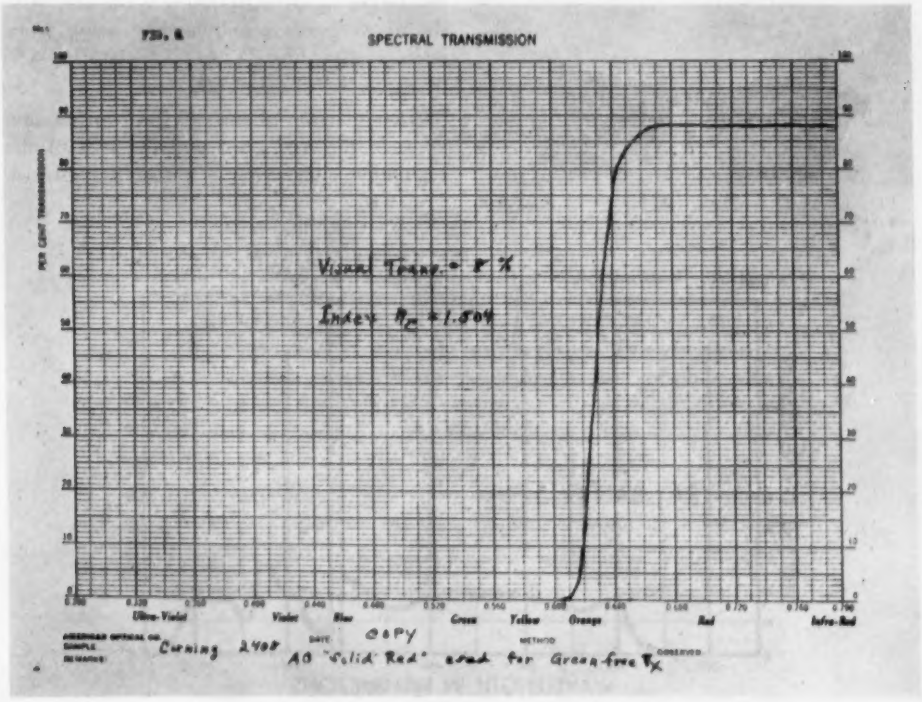

Figure 2

his fluoroscopy was performed in the morning shortly upon arrival at the office. During the summer there was an eight-mile drive in the sun from his home to the office, and he found that it took doubly long to dark-adapt his eyes, in spite of any sunglasses that were worn. The same problem also disturbed a radiologist. The usual procedure was to wear the regulation red plastic shield over their regular glasses and continue their other duties until dark-adapted. The internist complained that the shield was unsightly to his patients and that the glasses frequently steamed up.

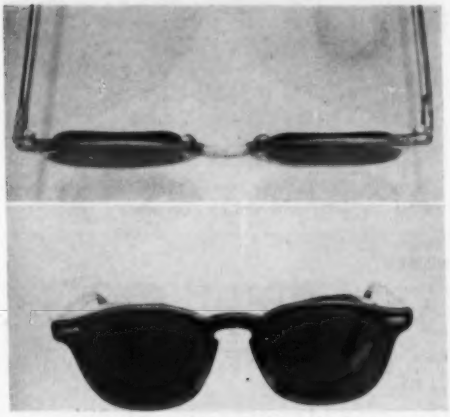

Fig. 3.—O. D. −10.50 −4.50 × 8; O. S. −7.50 −4.00 × 7.

The problems were solved by the use of a red glass, accurately ground to prescription and mounted in an appropriate frame. The lens used was the Corning Red Glass No. 2408.* This glass is known as the "sharp-cut" type, and, therefore, there is little change in transmission with a change of thickness. The visual transmission is 8%. The index of refraction is 1.504. The corresponding index of refraction for crown glass is 1.520 (Fig. 2). Therefore, if a prescription glass is made up in this glass, the curve should be strengthened approximately 0.03 D. per diopter.

The Corning Red Glass can be ground from plano to 4-5 D., plus or minus, without any difficulty. Prescriptions up to 10 or 20 D. require a process of thermobinding. Bifocals and trifocals can also be successfully made by the same process, resulting in an even density of color throughout the entire lens.

A correction for a gastroenterologist in the following prescription was possible with the above process and is now being worn successfully (Fig. 3): O. D. −10.50 −4.50 × 8: O. S. −7.50 −4.00 × 7.

A comparison of the spectral transmission between this red glass and the best of the regulation sunglasses is noted in Figure 4.

* American Optical Company, Southbridge, Mass.

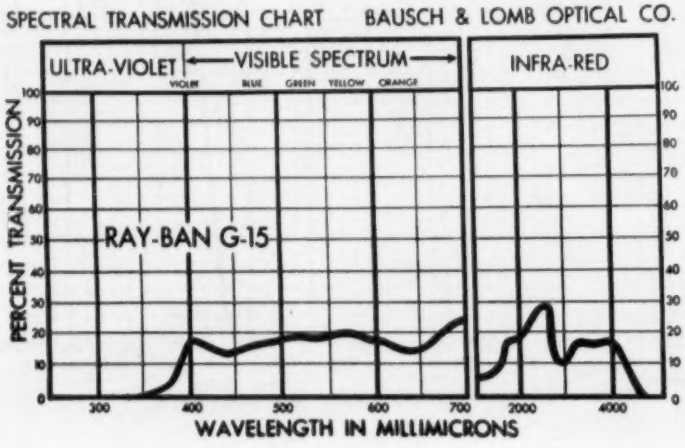

Figure 4

Summary

The Corning Red Glass was found to be satisfactory for optical grinding in corrections from plano to 4-5 D., and, by a process of thermobinding, corrections up to 20 plus or minus, and bifocals or trifocals, were possible. This type of glass is eminently suited for persons using a darkroom and who must wear corrective lenses. The four patients who received these glasses have found them superior to the use of the regulation red plastic shield worn over their glasses. One patient has worn the single vision correction for four years and is now wearing the bifocal additions.

The lenses and frames were made by The House of Vision (Belgard-Spero Co.), Milwaukee.

238 W. Wisconsin Ave.

Annual Reviews

The Orbit

A Review of the Literature for 1956

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Since the last annual review of the orbit [1] there have been several interesting articles. Most of the articles are reports of interesting cases or reviews of specific problems with case reports.

Anatomy

An excellent report of the development of the orbital facia and the variations in the adult was presented by W. H. Fink.[2] In this investigation, sections of several embryos were studied and the manner of development of the orbital fascia was considered. There was also consideration of the various types of anomalies. Dr. Fink's interest in the matter of orbital fascia and the oblique muscles is well known, with this being a worthy contribution to his previous articles. The significance of fascia abnormalities and their importance in the production of strabismus is undoubtedly a controversial subject. The difficulty exists because one cannot prove the presence of abnormal innervation; therefore, until tests are devised to establish a neurogenic factor, the controversy of the mechanical and innervational production of strabismus will continue.

Diagnostic Methods and Instrumentation

The problem of establishing an etiological as well as anatomical diagnosis in orbital tumors continues to confront the ophthalmologist. Keskey and Letsch [3] report their findings in 17 cases of orbital masses by the use of retrobulbar air injection with planigraphy. It was possible to obtain a satisfactory examination in 12 cases, with correct localization in 9 cases. The authors' technique was to use a 24-guage 5 cm. short-beveled needle on a 50 cc. Luer-Lok syringe. The needle was inserted into the posterior muscle cone, and 20 to 30 cc. of air was injected rapidly. The necessity of producing a marked exophthalmos was stressed. Physical discomfort to the patient necessitated the use of general anesthesia, but there was minimal discomfort after the air had been injected. The conclusions were as follows: (1) The use of air delineates some space-occupying lesions transparent to x-ray; (2) the procedure provides an evaluation of the tumor volume and position, and (3) there was minimal discomfort. Psenner [4] published a good paper on the diagnosis of space-taking lesions of the orbit, with particular details on the x-ray findings. There were no new techniques discussed but rather a good review of the entire subject of orbital tumors.

Ellis and Levy [5] published a follow-up article concerning the use of a cone-shaped implant in the muscle cone following enucleation. The implant is completely buried and contains permanent magnets for increased motility. The basic principles involved appear sound.

Malone [6] reviewed his experiences in management of foreign bodies of the globe and orbit. The author stressed the importance of accurate localization, early surgery, and care in minimizing damage to tissue at the time of surgery. The author appears

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to agree that orbital foreign bodies need not be removed but that treatment for infection is important. In the management of intraocular foreign bodies the benefit of the Berman locator is stressed.

Naquin [7] described a new procedure used in one patient in which the temporal muscle was used to help fill the orbital defect following an exenteration. An opening was made in the lateral orbital wall, and through this opening a pedicle of the temporal muscle was placed in the orbital cavity. This is an ingenious technique, but in view of the size of the cavity following an exenteration and the limited amount of temporal muscle available, time will be required to evaluate just how much benefit is derived by such a procedure. Fuchs [8] briefly summarized a method of closing the defect at the end of an exenteration procedure. The technique was to bring the skin edges together and allow the defect to granulate in with the benefit of external pressure.

Quereau and Souders [9] reported on the use of polytetrafluoroethylene (Teflon) for plastic correction of depressed fractures of the orbit. The material Teflon has no known solvent, is chemically inert, and does not cause an inflammatory reaction. The material is unaffected by temperatures of 620 F and is easily cut with a knife. On the basis of this report, it would appear that Teflon may be of great value when tissue volume needs to be replaced, but only time will provide the answer of whether there may be late complications.

Congenital and Developmental Anomalies

The importance of myopia in the production of exophthalmos is well presented by Bertelsen.[10] The author studied this problem by the investigation of patients with a high degree of myopia in one eye. There were 36 patients studied with a refractive difference between the two eyes ranging from 3 to 16 D. The average difference of myopia between the two eyes was 7.8 D. One of the most interesting observations was that the amount of proptosis of the highly myopic eye was related to posterior segment changes. If there were myopic posterior segment changes, the anterior eye position was changed little if any. If the posterior segment was normal, the exophthalmos was more marked. The author stresses the importance of determining the refractive error of patients with unilateral exophthalmos.

Urrets-Zavalía [11] published a good review of developmental dysostosis of the skull and face. The author considered the various classifications and pointed out that a simple classification is not easy. He also observed that there may be a combination of malformations in many patients.

Bruwer and Kierland [12] published an excellent article on congenital unilateral exophthalmos. Five of the patients had dermatological evidence of neurofibromatosis. Three of the six cases had pulsating exophthalmos. Skull x-rays revealed the typical neurofibromatosis lesions. Not all of the cases had retrobulbar neurofibroma. This is a complete review of this problem.

Carreras and Galvez [13] presented a case of digito-ocular phenomenon of Franceschetti. This condition was first described in 1937 by Franceschetti. The diagnosis is made by the characteristic bone changes in the orbit and fingers.

Inflammation

Sebestyen [14] reported on the treatment of 50 cases of acute inflammation of the orbit with the sulfonamides and antibiotics. The author reports a reduction in blindness from 15% to 18% to 3% to 5% since the advent of sulfonamides and antibiotics in the treatment of acute orbital inflammation.

Cutler and Blatt [15] reviewed the literature and presented two cases of lethal midline granuloma (Wegener's granulomatosis). A total of 110 cases were found reported in the literature. Forty-two per cent of the patients had ocular involvement. Regardless of treatment the prognosis is very poor. As yet no etiological agent is known.

Linhart [16] reported a single case of lethal granuloma of the orbit. The author's case presented the characteristic malignant type of course but no demonstrable tumor. The typical course starts with a watery discharge from the nose with an ulcer in the nasal vestibule. The second stage varies from 10 to 18 months. The nasal ulcer develops a granular surface with induration. The process gradually extends to the palate, nasal, and malar bones, with necrosis. The third stage is characterized by exhaustion and extreme malnutrition, with death due to high temperature or bleeding. The use of surgery may make the condition worse. There is no known treatment.

Sarcoidosis of the orbit is a rare condition. Stein and Henderson [17] present the eighth known case in the literature and review the literature. There were only 2 cases of sarcoidosis of the orbit in 1000 primary orbital tumors reviewed at the Mayo Clinic. The value of therapy is difficult to evaluate because there may be spontaneous recovery. The condition is rarely diagnosed before a biopsy is performed.

Gruber [18] reported a case of bilateral swelling of the lacrimal gland with Sjögren's syndrome due to sarcoidosis of the lacrimal glands. The author brings up the question of whether sarcoidosis, Sjögren's syndrome, and Mikulicz's disease are related.

Paranasal Sinuses and Bone

An osteoma of the orbit in a 52-year-old man was reported by Wheeler.[19] The history was a gradual swelling of the left eye without pain. X-rays established the diagnosis, and surgical removal was successful. The tumor mass weighed 29 gm.

Leonitiasis ossea is a condition of unknown etiology in which there is massive overgrowth of the frontal bone. The condition causes proptosis, with displacement of the globe and diplopia. Unfortunately, there is usually a serious cosmetic defect as well as physical discomfort. Jain, Sethi, and Parkash [20] reported a single case.

Vascular Conditions

Whitwell [21] reported a case of a spontaneous hematoma of the orbit in a 63-year-old woman. The patient had a sudden headache, exophthalmos, chemosis, and dilated vessels of the conjunctiva. There was no lid discoloration. The vision was reduced to 6/24. Results of a complete physical examination were normal. At surgery blood was drained from the orbit, and there was complete recovery. Spontaneous hematoma of the orbit is rarely seen in the absence of general disease.

Two children with recurring exophthalmos who had hemangiomas removed in infancy were reported by Niessen.[22] The subsequent clinical picture was that of pseudotumor of the orbit.

Serfling and Parnitzhe [23] reported seven cases of arteriovenous fistula in the cavernous sinus observed 6 to 10 years. Six of the seven cases had been operated on, with improvement. The value of angiography in the diagnosis of the condition is stressed.

Manifestations of General Disease

Two unusual cases of leukemia with the presenting symptom of unilateral exophthalmos were reported by Kennedy and Green.[24] This report again presents proof of the value of blood studies in patients with exophthalmos. One of the patients had an intermittent exophthalmos, the mechanism not being proved.

There was an excellent symposium on exophthalmos in 1956. Henderson [25] reviewed the problem from the ophthalmologist's standpoint. He divided exophthalmos into four main groups: (1) inflammation, (2) vascular anomalies, (3) metabolic exophthalmos, and (4) orbital tumors. Rose [26] reviewed the endocrine aspects of exophthalmos. He does not believe that there are two types of endocrine exophthalmos (thyrotoxic and thyrotropic) but rather feels that there is a common etiology, with the different clinical pictures due to gradiations of the same process. Jefferies [27] con-

sidered the treatment of exophthalmos from the viewpoint of the internist. He reports that the four primary causes are (1) stress, (2) pituitary dysfunction, (3) thyroid dysfunction, and (4) increased orbital fluid. There is uniform agreement on increased orbital fluid but not on the mechanism. The mechanism is probably an increased deposition of mucopolysaccharide which has strong affinity for binding water. Corticotropin and cortisone do help. The general outline of therapy is (1) general supportive treatment, (2) local eye treatment, (3) management of thyroid disfunction, (4) corticotropin and hydrocortisone, (5) irridation, and (6) orbital decompression. Fry [28] reviewed exophthalmos due to orbital masses. His classification of exophthalmos was (1) inflammatory, (2) vascular, (3) trauma, (4) primary orbital tumors, (5) orbital condition secondary to primary disease elsewhere, and (6) metastatic involvement of the orbit.

Exophthalmos of Thyroid Disease

As usual, most of the articles on the orbit concerned exophthalmos secondary to thyroid disease.[29-42] The literature continues to contain divergent opinions and for the most part is a rearrangement of previously presented material.

Campbell and Tonks [29] presented interesting experimental work with rats. Their conclusions were as follows: "Within the limits of these experiments . . . thiouracil was found to cause a mild degree of exophthalmos, which was probably due to the uninhibited action of thyrotropic hormone in a state of depressed activity of the thyroid. Cortisone not only caused exophthalmos in rats when acting alone, but its immediate action was exacerbated in animals previously treated with thiouracil. From the clinical point of view it would appear that treatment with thiouracil is not a safeguard against the occurrence of exophthalmos, and that the latter may be exacerbated during treatment by exposure to stress."

The eyes and pituitary hormone were the subject of a discussion by Greene, Brain, Pennybacker, and Stallard.[30] This was a good survey of the problem, but there was no new material presented.

Otto [31] reported on seven patients with malignant exophthalmos. The patients were treated with vitamin A or intraorbital hyaluronidase, with no benefit. Irradiation of 3000 r to the orbit did not help, but surgical decompression was beneficial.

The use of cortisone and prednisone in a case of malignant exophthalmos appeared to be helpful. Remission occurred after 16 months of therapy. The case was reported by Leftwich.[32]

Rowbotham and Clarke [33] reported on 30 cases of orbital decompression (15 primary tumors, 15 cases of thyrotoxcosis). The operative procedure consisted of removal of the orbital roof and lateral wall. The dura of the anterior and middle cranial fossa were exposed. The surgical results were reported to be good.

A general review of exophthalmos of thyroid disease was presented by Fraser.[34] This article is academic and contains no new concepts.

In a letter to the editor, Cohen [35] stressed that one should prove whether thyroid exophthalmos is in the residual or evolutional stage. According to the author, one can use the thyrotropin level in body fluids to establish whether the patient is in the residual or evolutional stage.

One of the most interesting articles concerns the significance of hypercementosis in the thyroid patient and the development of exophthalmos. Kupfer [36] reports that hypercementosis (abnormal thickening of cementum of the teeth) is four times as frequent in hyperthyroid women without progressive exophthalmos as in normal or hyperthyroid women with exophthalmos. The absence of hypercementosis, then, increases the hazard of exophthalmos.

Haner [37] published an excellent paper on clinical observations of endocrine exophthalmos. The author agrees with others in that

the conception of thyrotropic and thyrotoxic exophthalmos is unnecessary. The endocrine exophthalmos and the degenerative and infiltrative changes in muscles and other tissues are essentially the same, regardless of thyroid activity. He also observed that there was a 1 to 3 mm. difference in the protrusion whether the subject was upright or reclining, in the normal subject. This change is also found in the patient with thyrotoxocosis unaccompanied by ophthalmoplegia. This change was not found in exophthalmos with ophthalmoplegia whether thyrotoxocosis was present or not. The presence or absence of the change in the exophthalmos depending upon the subject's position may be of value in treatment.

Laurent and Scopes [38] reported on a case of exophthalmic ophthalmoplegia treated with the retrobulbar injection of hyaluronidase. The patient was a 53-year-old man who had received radiation therapy with no benefit. The injections resulted in some reduction of the edema but had questionable benefit for the diplopia or exophthalmos.

McKean and Hamburg [39] presented a general review of the subject of medical aspects of exophthalmos. There was no new material presented in this paper. Ruedemann [40] presented a similar paper.

An excellent paper by Wagener [41] reviews the mechanism producing exophthalmos and the complications. The author places emphasis on the visual aspects of the problem. A similar paper by Rohrschneider [42] presents a German review of the problem of endocrine exophthalmos.

Tumors

A good review of the management of orbital tumors, especially of the lacrimal gland, was presented by Reese.[43] In his article there is a good presentation of the surgical approach to these tumors. An interesting case of a lacrimal gland tumor in a 24-year-old Negro man was reported by Arnold.[44] The patient had a history of a prominent eye from childhood, but he had a sudden change in the degree of exophthalmos, with some visual reduction. At operation a cyst of the lacrimal gland of walnut size was removed.

Milam and Heath [45] reported a review of primary tumors of the lacrimal gland from Boston over a 60-year period. There was a total of 37 cases. Twenty-nine of the tumors were of the epithelial portion of the lacrimal gland. Twelve, and possibly fifteen, of the patients died of the disease. The significance of this study is that tumors of the lacrimal gland should be considered clinically malignant, with almost 50% of the patients dying of the tumor. The deaths occurred in a period of a few months to 28 years.

Nicholls [46] reported three cases of cholesterol-containing granuloma of the orbital wall. The patients had had symptoms of exophthalmos from a few months to several years. Surgical removal was successful.

Two patients with unusual myogenic tumors around the eye were reported by Blodi.[47] One patient was a 36-year-old man with a history of a slow-growing tumor arising from the muscles of the orbital margin, with no bone defect. There was a history of previous trauma. Microscopic sections revealed the tumor to be a granular-cell myoblastoma. The second patient was a 7-year-old child with an embryonal rhabdomyosarcoma. There were 20 cases of rhabdomyosarcoma reported in the literature (ages 2 to 9 years, average age of onset 6 to 7 years). In children this is the commonest primary malignancy of the orbit.

A 41-year-old woman with a painless swelling of the orbit was reported by Skeoch.[48] The encapsulated mass was excised and proved to be a neurilemmoma of the orbit. The clinical diagnosis was a dermoid tumor of the orbit.

A case of eosinophilic granuloma of the orbit cured by excision and curettage was reported by de Queiroz and Barbi.[49] The authors recommend radiation therapy following surgery. Kurus [50] reported a patient with giant-follicular lymphoblastoma

of the orbit cured by surgery and radiation therapy. The author recommends surgical excision followed by x-ray treatment.

A 50-year-old male patient with proptosis from multiple myelomatosis was reported by Handousa.[51] The patient had the typical bone changes and positive Bence Jones protein.

An excellent paper on conservation of the eyeball in the treatment of certain tumors of the orbit was published by Jackson.[52] The author reported two patients with glioma of the optic nerve in whom the nerve was removed but the globe was left intact. Both patients are still alive (one 18 years, one 20 years). Two patients with sarcoma both died following surgery and x-ray therapy. Comments by Jackson are as follows:

"Although this series is of limited number, there are a number of points of especial interest to be observed.

1. The benefit of avoiding loss of the eye when dealing with glioma of the optic nerve.

2. The feasibility of treating tumours of the optic nerve and malignant tumours of the orbit without removal of the eye.

3. The occurrence of primary malignant tumours of sarcomatous nature within the orbit in two quite different age groups:

(*a*) Children in whom a short history and poor prognosis are particular features.

(*b*) Adults over 65 years in whom the history of the disorder is usually longer and the progress not so sinister.

4. The disadvantages of radical surgical interventions as indicated in the form of extirpation and in the manner of approach:

(*a*) Exenteration seems to afford little prospect of cure, and exposes the patient to the likelihood of formation of an unpleasant and unsightly fungating mass.

(*b*) Operative measures whereby tissue spaces are widely exposed may encourage implantations of neoplasm in the track of the wound.

5. Deep x-ray therapy does not of necessity mean destruction of the eye, either functional or structural, during the course of the attendant disease.

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News and Comment

ANNOUNCEMENTS

University of Colorado Postgraduate Course in Ophthalmology.—The Colorado Ophthalmological Society and the Office of Postgraduate Medical Education, University of Colorado School of Medicine, are co-sponsors of a Postgraduate Course in Ophthalmology to be given July 22-25, 1957, at the University of Colorado School of Medicine, concurrently with the Summer Convention of the Colorado Ophthalmological Society.

The four-day course will consist of a symposium on glaucoma and lectures on refraction and optical aids in reduced vision. Lecturers for the symposium will be Drs. Robert N. Shaffer, Robert A. Schimek, and H. Saul Sugar. Dr. Arthur Linksz will conduct the other lectures.

Registration is open to all members of the Colorado Ophthalmological Society and other qualified physicians on a limited membership basis. The registration fee is $5.00, and the tuition fee is $45.00.

Applications, together with the $50.00 fee, should be sent to the University of Colorado Medical Center, Office of Postgraduate Medical Education, 4200 E. 9th Ave., Denver 20.

Pan-Pacific Surgical Association: Seventh Congress.—The Seventh Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Hawaii, Nov. 14-22, 1957. All members of the profession are cordially invited to attend and are urged to make arrangements as soon as possible if they wish to be assured of adequate facilities.

An outstanding scientific program by the leading surgeons with sessions in all divisions of surgery and related fields promises to be of interest to all doctors.

Further information and brochures may be obtained by writing to Dr. F. J. Pinkerton, Director General of the Pan-Pacific Surgical Association, Room 230, Young Building, Honolulu, Hawaii.

Committee on Industrial Ophthalmology.—The Council on Industrial Health, American Medical Association, is pleased to announce the completion of its new Committee on Industrial Ophthalmology. Members of the Committee are Edmund B. Spaeth, M.D., Chairman, Philadelphia; Edmund L. Cooper, M.D., Detroit; Franklin M. Foote, M.D., New York; Ralph S. McLaughlin, M.D., Laconia, N. H.; Joseph F. Novak, M.D., Pittsburgh; Ralph W. Ryan, M.D., Morgantown, West Va.

The first meeting of the Committee was held in New York, June 7, in conjunction with the annual convention of the American Medical Association.

Seminar on Glaucoma.—A Seminar on Glaucoma with particular emphasis on gonioscopy and the study of the anterior angle will be given at the Brooklyn Eye and Ear Hospital on Nov. 18, 19, and 20, 1957. Ample opportunity for practical instruction in the use of the gonioprism will be given, and material from the glaucoma clinic will be utilized.

The course will be given by Dr. Daniel Kravitz, assisted by Drs. Mortimer A. Lasky, A. Benedict Rizzuti, Abner S. Rosenberg, Nicholas P. Tantillo, and Samuel Zane.

Registration is limited to six ophthalmologists only.

Application and the fee of $50 may be addressed to Dr. Daniel Kravitz, Brooklyn Eye and Ear Hospital, 29 Greene Ave., Brooklyn 38.

PERSONAL

Mr. A. J. B. Goldsmith.—Mr. A. J. B. Goldsmith, F. R. C. S., has been appointed visiting professor of ophthalmology at the University of Toronto for the month of May, 1957, as exchange lecturer from the Middlesex Hospital, London, England.

Dr. Edward J. Donnelly.—Dr. Edward J. Donnelly has been appointed attending surgeon at Wills Eye Hospital, Philadelphia. He has been a member of the staff for 25 years. He is also active consultant in Ophthalmology at St. Vincent's Hospital and at Philadelphia General Hospital and assistant professor of clinical ophthalmology at Jefferson Medical College.

Dr. Donnelly is an alumnus of St. Joseph's College and received his medical degree from Jefferson Medical College. He is a specialist certified by the American Board of Ophthalmology; a fellow of the American Academy of Ophthalmology and Otolaryngology, the American College of Surgeons, the International College of Surgeons, the College of Physicians of Philadelphia, and the Pennsylvania Academy of Ophthalmology and Otolaryngology, and a member of the American Medical Association, the Philadelphia County Medical Society, the Ophthalmic Club of Philadelphia, and the Medical Club of Philadelphia.

SOCIETY NEWS

Section on Ophthalmology—Annual Scientific Session.—The Annual Scientific Session of the Section on Ophthalmology, American Medical Association, was held in New York from June 5 to 7, 1957. An audience of 500 persons was in attendance throughout the meeting.

Distinguished foreign guests were Prof. J. François, Ghent, Belgium, and Mr. Frank Law, London, England.

Several prizes and honors were awarded:

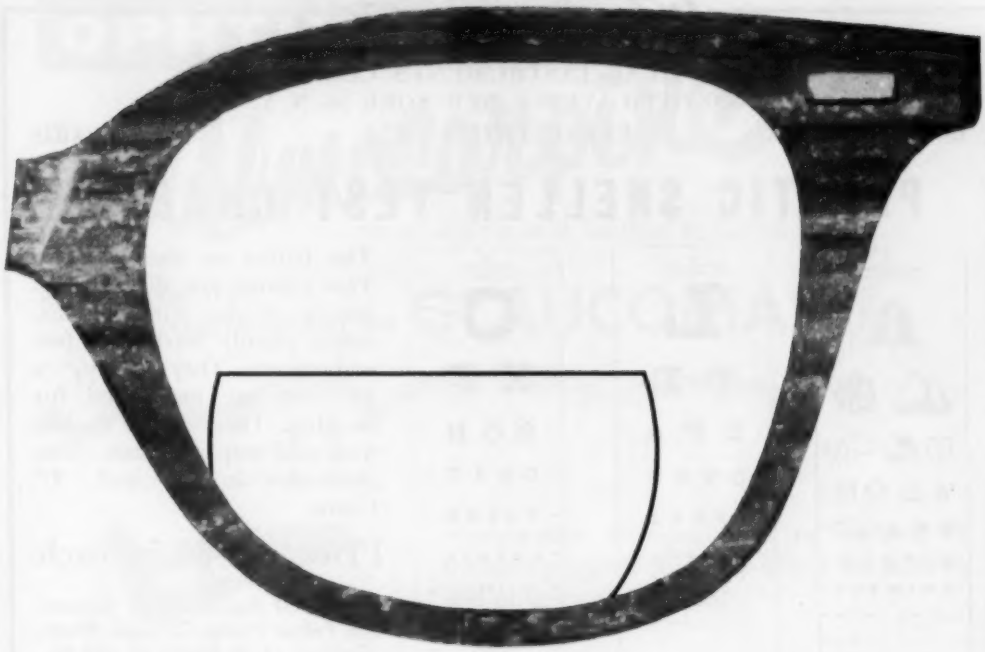
1. The Knapp Medal was awarded to Goodwin M. Breinin and Joseph Moldaver for their paper "Electromyography of the Extraocular Muscles," which was presented before the Section in 1955.

2. The Prize Medal in Ophthalmology was awarded to William L. Benedict.

3. The $250 prize for the best exhibit was awarded to David Volk for his exhibit "Conoid Ophthalmic Lenses: An Original Development in Subnormal Visual Aids."

4. The $250 prize was awarded to James E. Bennett and Arby L. Bailey for their paper entitled: "A Surgical Approach to Total Xerophthalmia Through Transplantation of the Parotid Duct," judged on the basis of presentation and originality.

The following officers were elected: Dohrmann K. Pischel, Chairman; Ralph W. Danielson, Vice-Chairman; Harold G. Scheie, Secretary; Ralph O. Rychener, Delegate to House of Delegates; Frank W. Newell, Representative to Scientific Exhibit.



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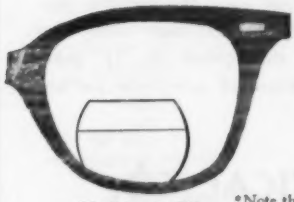

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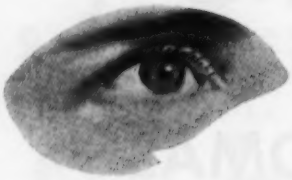

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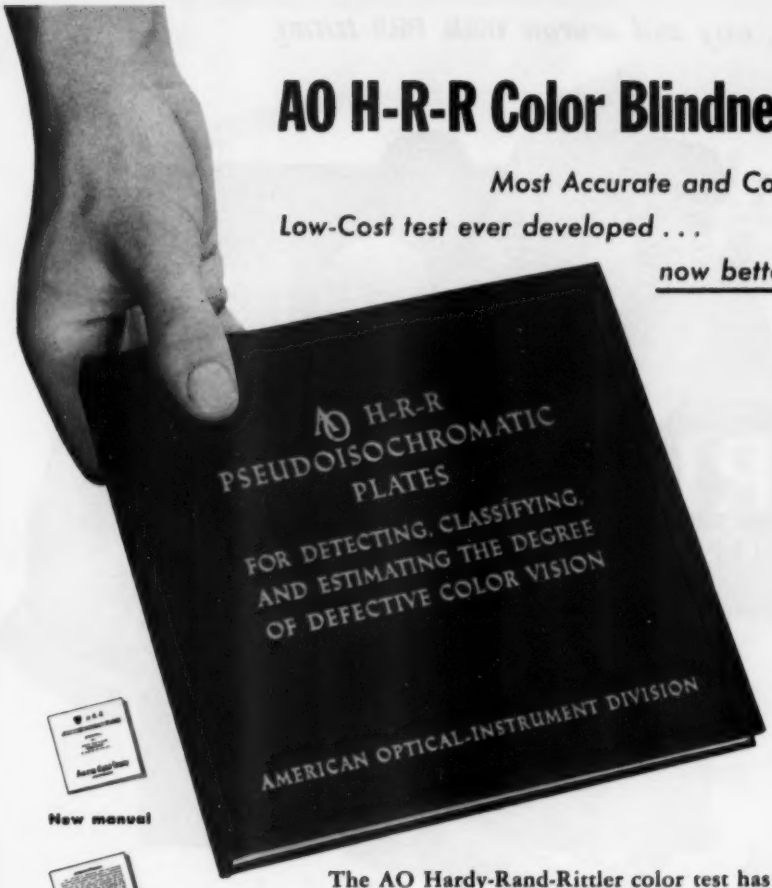

New manual

New instructions

New scoring sheets

New order of presentation

The AO Hardy-Rand-Rittler color test has answered the need for an easy-to-give, comprehensive, low cost color blindness test. It is the result of more than ten years of scientific investigation, development and validation by eminent optical authorities.

The test not only detects people who have Red-Green and/or Blue Yellow color blindness but also types the deficiency and estimates the degree of defective color vision present.

The new improved Second Edition takes advantage of several years of field experience. Now, the new manual, instructions, scoring sheets and order of presentation make the test even easier for the practioner to give and easier for the patient to understand. The total content of the test remains unchanged. The universally understood recognition symbols remain the same; a circle, cross and triangle. The ingenious pattern of the plates allow no clues for memorization.

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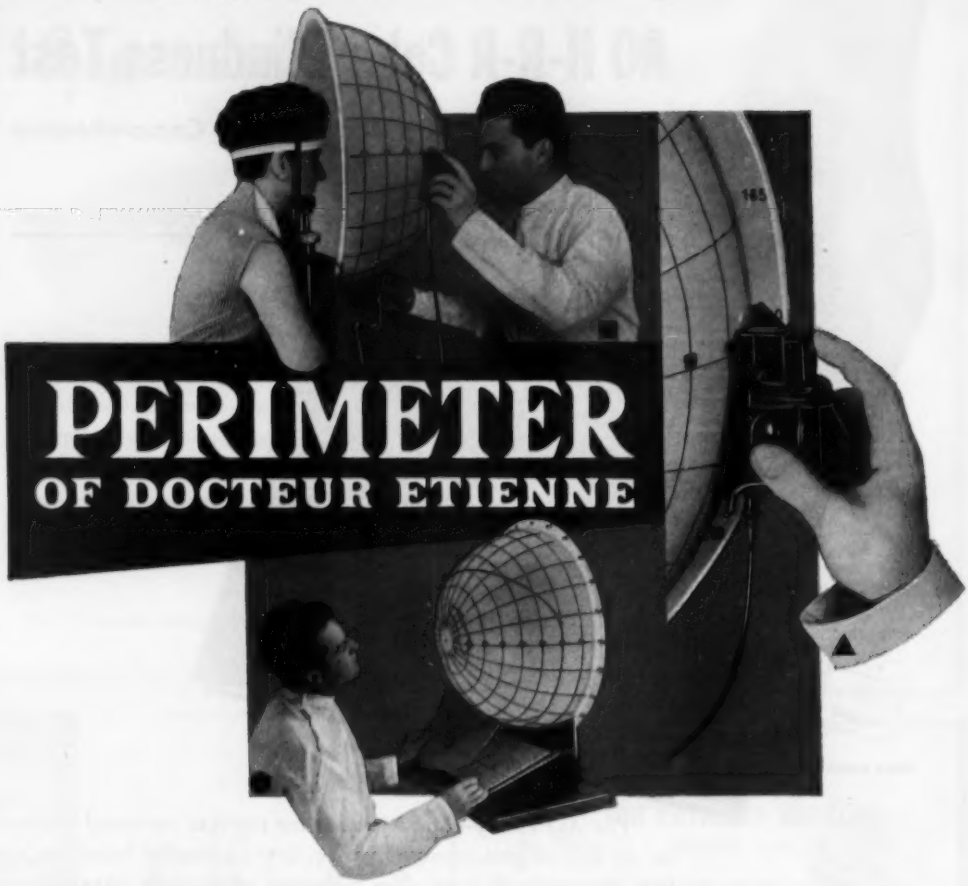

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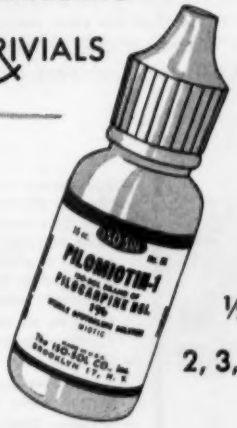

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University of California School of Medicine

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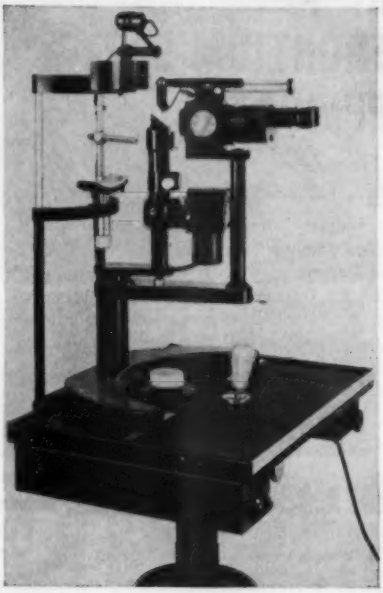

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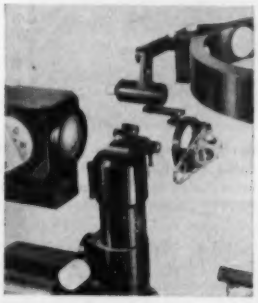
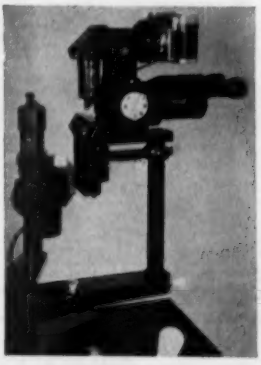


ATTACHMENT FOR GONIOSCOPY (left) consisting of: 4-mirror contact glass with handle, carrier and rotating device. Rota-table prism in mount. To keep lid apart during examination we offer one each small and large lid separator.

PHOTO ATTACHMENT (right) with electronic flash equipment to automatically photograph anterior segment of eye at magnifications of 1x or 2x.



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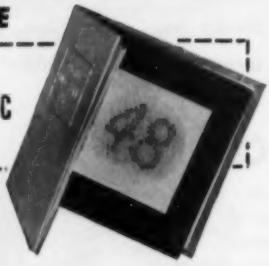

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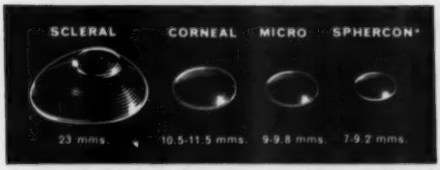

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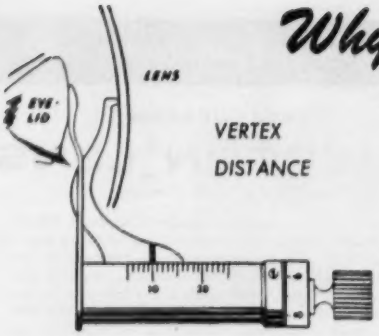

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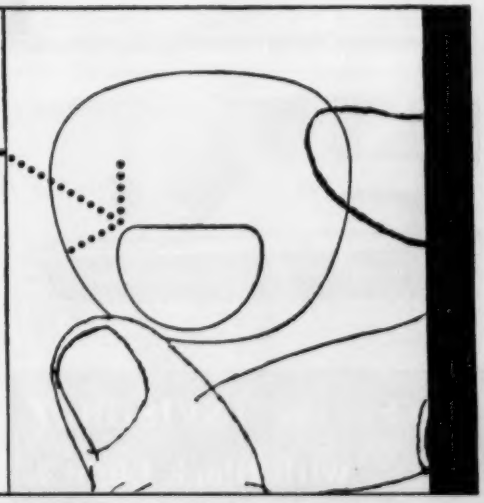

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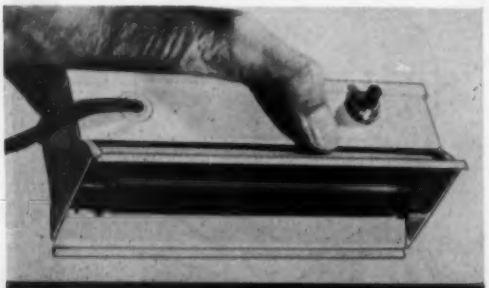

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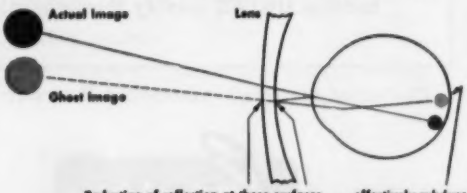

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Case History #3 explains the usefulness of Catarex Service in cases of bilateral cataract extractions. In this case, immediate vision was especially important, since the patient, a diabetic, lived alone and had to administer insulin to herself daily.

This case history emphasizes the fact that CATAREX T temporary cataract bifocals have proved themselves more versatile in aphakic cases *than any other form of temporary lens.* CATAREX T lenses are a simple answer to the old, complicated problem of easing the patient through the post-operative period.

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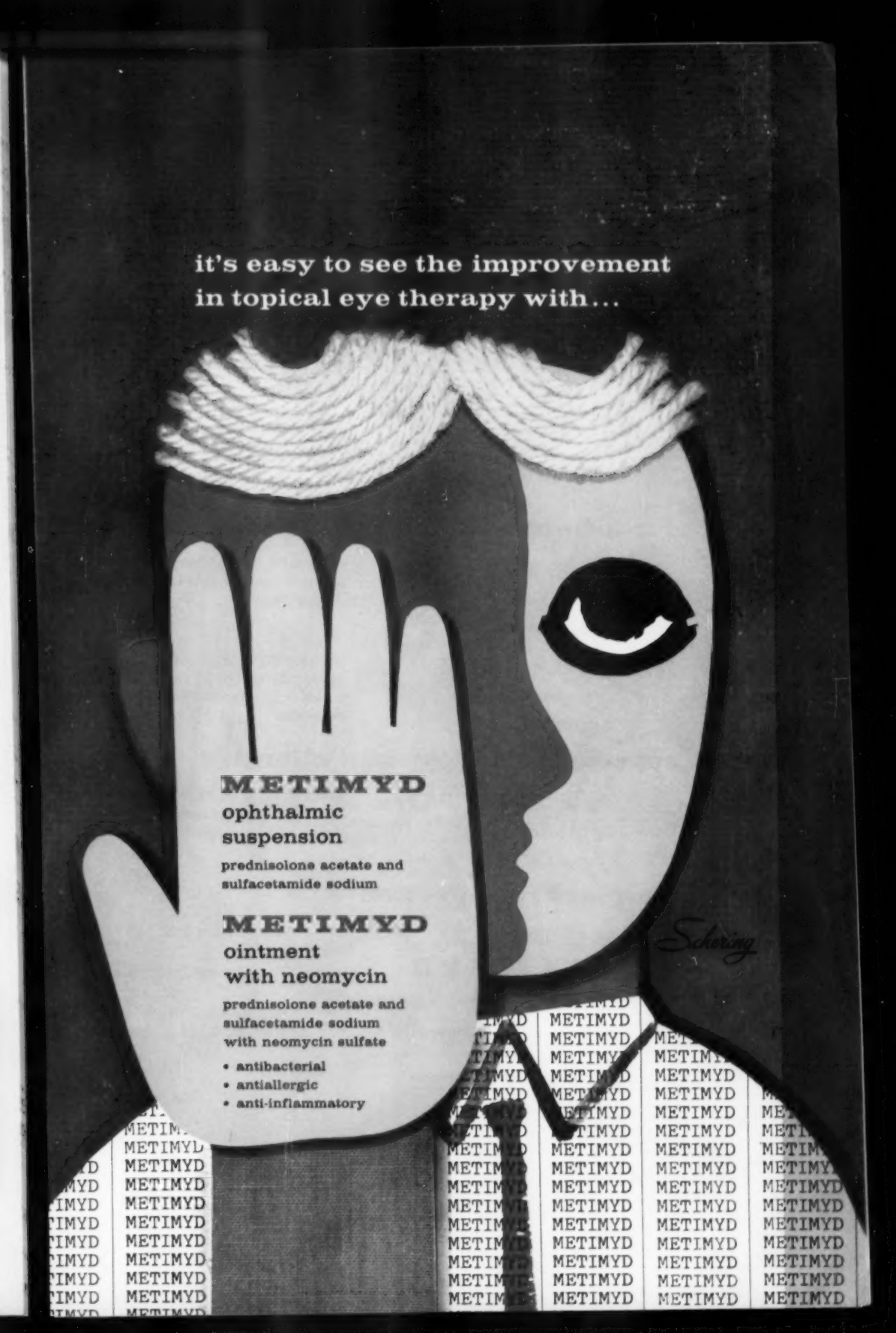
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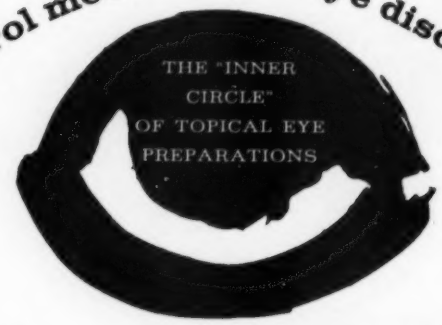

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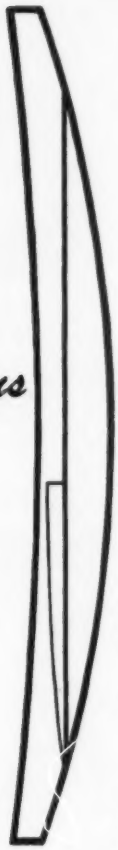

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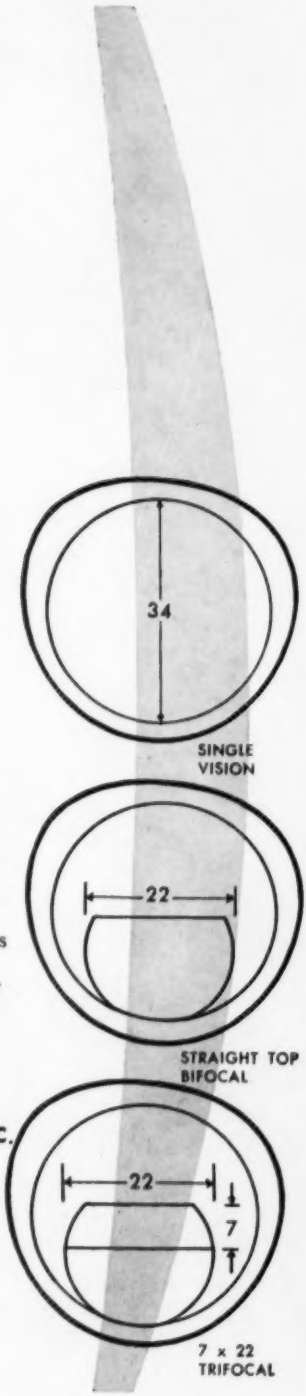

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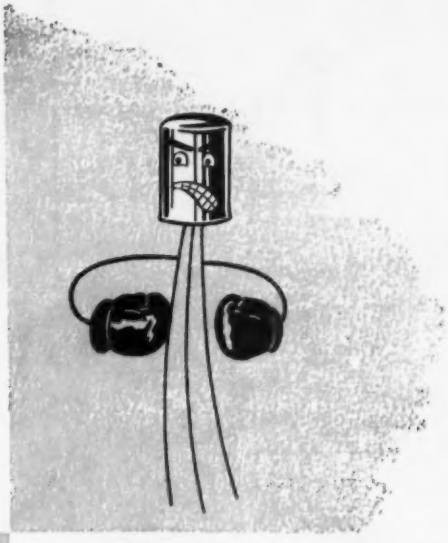
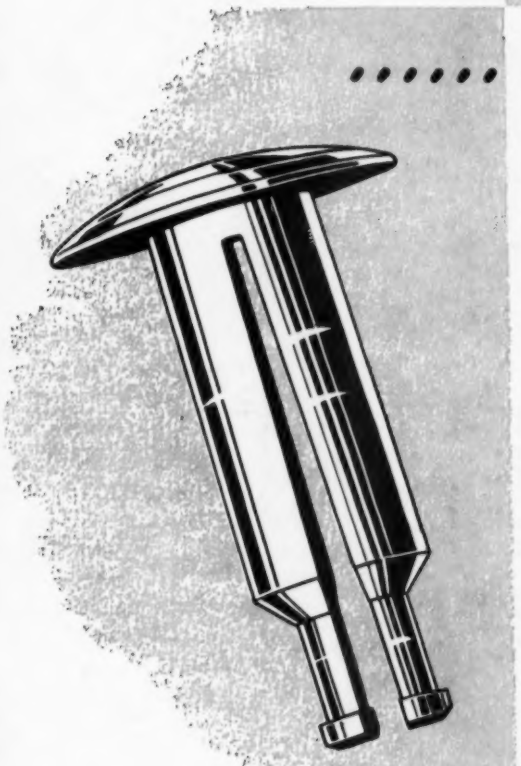


..... *so does*

TEMPELOC



Tempeloc is Continental's new 5-barrel hinge pin. It replaces temple screws. What a wonderful improvement you'll find it to be. Tempeloc is a small spring steel split pin, simple and quick to use. Insert it in the temple joint screw hole and push down with a plier or the flat of a screw-driver. It snaps in, spreads out, and locks itself securely. *The click's the clue* that it's tightly fastened.

With Tempeloc there's no screw backout, no temple fall off. Less laboratory time is required to insert Tempeloc pins than screws, and pins can be quickly pushed up and out in case of repairs or replacement.

Try Tempeloc. The pins fit any 5-barrel hinge. Your independent jobber can furnish them. They come on all Continental 5-barrel frames at no extra cost.

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